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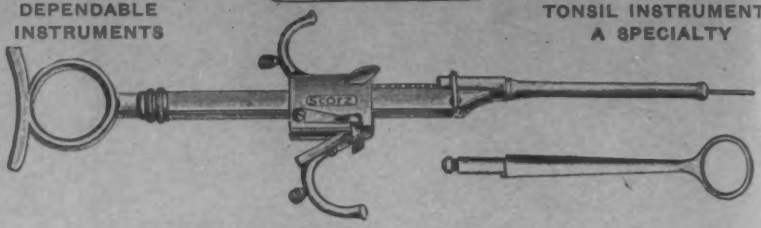

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Described in The Laryngoscope, Oct., 1926. Send for reprints.

CHAS. R. STORZ

3466 CARROLLTON AVE.,
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THE
LARYNGOSCOPE.

VOL. XXXVIII MAY, 1928. No. 5

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

NEW HISTOPATHOLOGICAL FINDINGS IN THE EAR IN LUES AND THEIR IMPORTANCE IN THE GENERAL PATHOLOGY OF THE EAR.*

DR. GUSTAV ALEXANDER, Vienna.

In the early studies of the histopathology of the ear, it was believed that the many characteristic findings for the various congenital and acquired ear diseases were known. With the increase of our knowledge, however, we became less certain as to the histopathological findings specific for congenital or acquired ear diseases; findings that were believed to be characteristic for congenital ear diseases (false formations and encapsulations in the inner ear, membranous duplications, etc) were found also in cases of acquired diseases. It was found possible also to produce these changes experimentally by causing inflammation of the inner ear. Defects of the scala septa considered at first to be characteristic for congenital deafness were later found also in certain cases of internal ear conditions not associated with deafness (Mayer, Neumann, Brunner, Nager), and in cases of internal ear diseases combined with otosclerosis (Brunner). It was also found that some changes seen in otosclerosis may exist in cases of congenital deafness as well. These findings lead me to believe that basal changes in cases of otosclerosis may be really congenital.

A number of changes which were believed to be pathological were found to be artificially produced in preparing a specimen. On the other hand, certain changes which were believed to be artificial were

*Read by invitation before the New York Academy of Medicine, Section of Otology, Nov. 11, 1927.

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demonstrated to be actual changes. For instance, Nager and others believed that the corpora amylacea found in the eighth nerve were artificial products; whereas Obersteiner and I showed that this is not so.

Some exudates in the endolymphatic spaces were commonly believed to be artificial changes; but Brunner saw such changes regularly and could produce them regularly in experimentally produced otitis interna vasomotoria. More and more on the basis of all the knowledge we possess we have tried to find out the changes in the ear so far as they may be characteristic for special forms of disease.

The great value of such histopathological examinations is shown in cases in which we were able to examine the ear one or more times

Fig. 1. Old lues. Otosclerotic foci in cochlea capsula (a, b, c).

during the life of the patients. In this direction we reached the following results: We found characteristic changes in the bone of the inner ear capsule in otosclerosis. Instead of concentric lamellar construction we found reticulated bony tissue; the old bone became absorbed and new bone formed in network; instead of small capillaries in the bone, we found large blood vessels. The end-result may be rarefaction of bone with formation of connective tissue scars in the spaces. These changes are going on in different regions of the ear during the entire lifetime of the patient without reaching any definite end-stage. We believed that to an important degree the defective hearing in otosclerosis may be caused by the changes in the inner capsule alone, as well as by degeneration of the nerve of the cochlear apparatus.

Characteristic findings were also found in Paget's disease (O. Mayer, Nager, Brunner, J. Fischer), as an ectasia of the pars inferior of the inner ear (Saccus endolymphaticus, Ductus reuniens, and ductus cochlearis). No Paget patient has a normal hearing. In these patients the ear disease may be caused by a stretching of the eighth nerve, by changes of the bone in the region of the internal meatus, and by changes in the endolymphatic sac and vestibular aqueductus.

The third characteristic histopathological findings were made out in cretinism. In Cretins we found fat tissue filling the round window and other regions of the middle ear, fat marrow in the bone, sometimes combined with otosclerotic changes in the inner ear capsule and atrophy of the nerve apparatus of the internal ear.

The fourth change noted is the sum of the changes forming the choked labyrinth in cases of brain tumor. For such investigations

Fig. 2. Old lues. Otosclerotic foci (a, b) pressing towards the tip of the cochlea (a) and the scala tympani of the vestibular part (b).

it was necessary to select cases in which the brain tumor was situated far from the ear—in the middle or anterior brain—so that we might be sure that this choked labyrinth was caused indirectly by the brain neoplasm or brain pressure or by other forces, wholly analogous to the development of the ophthalmologic choked disc.

One of the most interesting diseases in this connection is lues. The details of the clinical ear findings in these cases, as shown by otoscopical and functional tests, are known to be very important and interesting. I have the honor to demonstrate to you tonight the histological changes of the ear which I found in six cases of acquired lues. All of these patients were examined during their lifetime.

The first case was that of an officer, age 55 years, whose ears I repeatedly examined during life. His hearing was reduced on both sides to words loudly spoken near the ear. Histologically I found a chronic ostitis of the inner ear capsule in the form of a gumma (Figs. 1 and 3).

Fig. 3. Old lues. Cut through the basal coil (St1=Scala tympani, Sv1=Scala vestibuli) of the cochlea with three otosclerotic foci (o, o1, o2).

Fig. 4. Old lues. Cut through the basal turn of the cochlea with two otosclerotic foci (a, b).

Fig. 5. Old lues. Promontorium. Communication of the pathological blood vessels in the otosclerotic focus (b) with the mucous layer of the tympanum (a).

The changes on both sides are equal. In the specimen there are islands of three localizations: in the cochlear capsule is a gumma (Figs. 4 and 5) like otosclerosis; the tip of the cochlea is a little depressed by one of these islands (Fig. 2), the scali tympani of the base is compressed (Fig. 2, b); the stapes (Fig. 7) is ankylosed by connective tissue; the cochlear window and the cochlear aqueduct (Fig. 8) are obliterated; there is fat marrow in the temporal bone; and chronic exudate in the cochlea, as in cases of otitis interna vasomotoria (Brunner; Fig. 10). The changes were nearly the same as those characteristic of otosclerosis; three islands of such a gumma

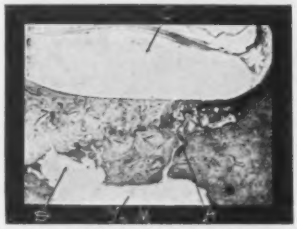

Fig. 6. Old lues. Cut through the base of the cochlea (c) and the internal auditorial meatus (Ta M). Perforating blood vessel (A) and defect of bone (D).

Fig. 7. Old lues. Stapes ankylosis (Sta) and obliteration of the aqueductus cochlea (Ac).

were present in the cochlear capsule, two on the base of the cochlea, one on the tip of the cochlea and pressing the tip a little downward. I saw these changes microscopically as I had the specimen in the microtome. The stapes was fixed by connective tissue. In the region of the windows, there were no changes in the bone cells. The cochlear aqueduct was obliterated by connective tissue (Fig. 8). It may be that this tissue came from the arachnoidea. On the cochlea, in the scala tympani and in the obliterating tissue were found sand bodies and corpora amylacea (Fig. 8). The superficial layers of the bone in the region of the internal auditory meatus showed cir-

cumscribed bone defects (Fig. 6), such as are found in cases of choked labyrinth due to brain tumors. In the mebranous cochlear canal, and in the tensor tympani muscle were some exudates and large quantities of bodies looking like corpora amylacea. The cochlear nerve apparatus (Corti's organ, spiral ganglion, and cochlear nerve) was found to be atrophic in the vestibular part of the cochlea (Fig. 9). In all other regions of the cochlea the nerve apparatus was found to be perfectly normal. This is very important, because these findings show that the very poor hearing this patient had was caused only by the changes in the bone. It may be that he was pre-

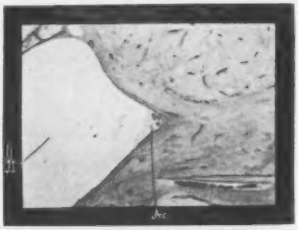

Fig. 8. Old lues. Scala tympani (St) with obliteration and sand bodies (Ac) on the cochlear aqueductus.

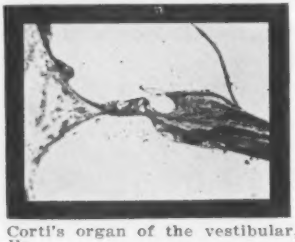

Fig. 9. Old lues. Corti's organ of the vestibular part of the cochlea with defective hair cells.

vented from hearing better because of the formation of such large blood vessels.

The specimens of the second case were presented to me by Prof. Dr. O. Mayer in Vienna. He saw this case during the lifetime of the patient and found him to be a man of about 63 years of age, with old lues and very poor hearing. Histologically, I found very greatly progressed ectasia of the inferior parts of the inner ear (Figs. 13 and 14), such as is found in Paget's disease, and very dense arachnoidal network in the cochlea aqueductus (Fig. 15); the formation of a membranous sac; and also osteophytes in the vestibule. The ectasia of the sacculus (Fig. 14), the canalis reuniens, and of the mem-

branous cochlea (Figs. 12 and 15) is tremendous. The neuroepithelium was found to be atrophic.

There is much atrophy of Corti's organ and of the stria (Figs. 11 and 12), with the formation of cysts and mounds and nets of connective tissue. There is a maximum amount of ectasia of the memduplication of its walls. Also, there are adhesions by connective branous cochlea, with duplication of its walls. Also, there are adhesions by connective tissue bridges (Figs. 11 and 13); superficial absorption in the petrous bone; and the dura of the internal meatus is chronically thickened. There are many sand bodies. The cochlear

Fig. 10. Old lues. Amyloid bodies and exudate in the cochlear canal on the tip of the cochlea.

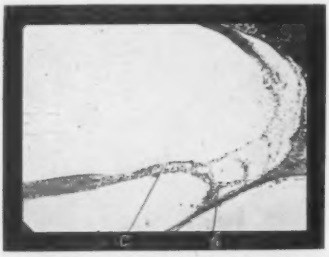

Fig. 11. Old lues. Cochlear canal with atrophy of Corti's organ (C). Connective tissue in the scala tympani (b). Specimen of Prof. Dr. O. Mayer.

aqueductus is permeable (Fig. 15). There are osteophytes in the scala tympani and in the vestibule (Fig. 14), with the formation of a vestibulocochlear septum (Fig. 14). There is maximal ectasia of the sacculus and ductus reuniens, with multiple cysts in the membranous walls. There are multiple pathological membranous sacs in the vestibule behind the stapes, osteoid tissue, connective tissue, and osteophytes. The annular ligament of the stapes is thickened. In the middle ear are connective tissue adhesions.

In the third case of lues examined clinically and histologically by my assistant, Hans Brunner, and published in his paper on Oto-

sclerosis (Monatsschrift fur Ohrenheilkunde, 1926), fat tissue was found in the middle ear, especially in the attic and in the petrous bone, combined with otosclerotic changes in the inner ear capsule.

In Brunner's case, the eighth nerve and its ganglion, as well as Corti's organ are atrophic. The stria is narrowed and is without blood vessels. The cochlear aqueduct is permeable. The attic is filled with fat.

In the fourth case, histologically examined by my assistant, Joseph Fischer, the findings were: nerve degeneration in the internal ear

Fig. 12. Old lues. Septum (S) on helikotrema with ectasia (E) of the cochlear canal. Specimen of Prof. Dr. Otto Mayer.

Fig. 13. Old lues. Ectasia (E) of the cochlear canal and connective tissue ligament (L) in the scala vestibuli. Specimen of Prof. Dr. Otto Mayer.

combined with pathologically intensive pigmentation and some perilymphatic and endolymphatic exsudate and obliteration of the cochlear aqueductus.

In this case a moderate degree of atrophy was found in the region of the cochlearis and small cysts in the entire extent of the labyrinth, especially in the region of the cristae ampullares. In the petrous bone were large pneumatic spaces and also fat marrow. The cochlear aqueduct is permeable. The entire inner ear is pathologically pigmented. In addition to the pigment bodies in the perilymphatic cells there are

two large areas of free deep-brown pigment. The folds are thickened, as in a chronic adhesive process.

Cases 5 and 6 were examined by myself. I found a moderate degree of atrophy of the internal ear, combined with obliteration of the cochlear aqueductus and changes in the internal auditory meatus, as in choked labyrinth caused by brain tumor.

In Case 5, the changes in both ears are practically equal. The meninges in the ear region are intensely thickened by connective tissue, especially the dural tissue in the left internal meatus. On both sides is a large amount of interglobular cartilage. The eighth nerve and its ganglion are nearly normal. In the vestibular part of the cochlear is a low degree of atrophy. The stria is narrowed and mostly very poor in blood vessels. In the tip of the cochlea are cysts with colloid content; similar cysts are found in both labyrinths, in the region of the utricle, and the semicircular canal. Corti's organ

Fig. 14. Old lues. Ectasia of the sacculus (S), vestibulo-cochlear septum (Sep) in the vestibuli. Specimen of Prof. Dr. Otto Mayer.

is mostly normal, but in some regions is atrophic. The cochlear aqueduct is barely permeable and is filled with arachnoid tissue. On the left side there is ankylosis of the malleoincudal joint.

I wish now to discuss the results of the findings, after comparing the histological findings in these cases of lues with the histological findings in the other diseases which I have already mentioned.

We have to see the changes found in lues are similar in many ways to the changes in the inner ear caused by other diseases. I mention here first the content of fat tissue in the inner ear already observed in cretinism and now found also in lues. It may be that this fat tissue really represents the low resistance of the organ which we call in German "minderwertigkeit". This may explain how in some cases of lues the ear is affected and not in others.

The finding of different degrees of atrophy in Corti's organ and in the nerve ganglion apparatus in many cases of lues is equal to the

changes in the inner ear typically caused by arteriosclerosis. We know clinically and anatomically that the internal auditory artery becomes changed very early, if at the beginning of the arteriosclerosis, the symptoms are otalgia angiosclerotica (Stein), bad hearing and noises. The reason for the ear being affected so early must be seen in the fact that the internal auditory artery runs isolated and branchless, as do most of the thinnest arteries in the human body, nearly the length of 30 to 40 m.m., and then divided into endarteries. Such an artery reacts very quickly to all kinds of direct and indirect injuries—therefore, also to arteriosclerosis. Therefore, one manner in which lues causes atrophy of the inner ear can be through endarteritis luetica.

The findings of ectasia of the pars inferior of the inner ear and the sac formation in the vestibule in our case of lues are the same as

Fig. 15. Old lues. Vestibulo-cochlear septum (Sep). Atrophy (A) of Corti's organ. Specimen of Prof. Dr. Otto Mayer.

those found regularly in cases of Paget's disease. The cause of the ectasia must be a change in the endolymphatic sac and duct (aquaeductus vestibuli) in the case of lues—as represented by luetic meningitis.

The findings of the complete or incomplete closure of the cochlear aqueduct in our cases of lues are the same as those found in a choked labyrinth caused by brain tumor. Any case of lues in the cochlear aqueduct is therefore the end-result of the luetic meningitis, pachymeningitis and encephalitis.

Of both practical and theoretical value is the finding of gumma in the inner ear capsule in cases of lues, resembling the changes in otosclerosis. These findings demonstrate that the opinion that otosclerosis may be caused by lues is entitled to consideration; and this

finding gives me a base for saying that *1.* in the future I shall use antiluetic treatment with neosalvarsan and hydrargyrum, and also the malaria treatment of Wagner in cases of otosclerosis. *2.* It is suggestive that antiluetic treatment is indicated as a prophylactic measure in children with a positive heredity of otosclerosis through parents or other near-blood relations. *3.* Also as a treatment for pregnant women with otosclerosis, in order to prevent the progress of the ear disease during the pregnancy. It may also be that this treatment may prevent in the developing embryo the formation of an ear organ of low resistance. *4.* It is necessary, too, that in cases of otosclerosis the antiluetic treatment should be given in the earliest possible stage after the disease has become manifest.

I have to mention still another point of physiological importance. In Case 1 the distance for hearing was very much diminished. The patient heard only very loud words spoken directly into the ear. An old experience has shown that such limited hearing cannot be caused alone by changes in the conductive apparatus but must also be combined with changes in the perception or nervous apparatus of the cochlea.

The histological findings in these cases show only a very low degree of nerve atrophy and atrophy of Corti's organ only in the beginning of the vestibular part of the cochlea. In all other ways, Corti's organ was perfectly normal from the beginning of the basal coil to the tip. The same normal findings showed in the cochlear nerve and spiral ganglion. We can make this exact histological diagnosis because the preservation of the structure of the inner ear was absolutely perfect.

This finding demonstrates that the great diminution of hearing in this case was caused only by the changes in the bone capsule, and allows the generalization that also extremely poor hearing can be caused by changes in the conductive apparatus alone, with no changes in Corti's organ and in the nerve ganglion apparatus.

CONSTITUTIONAL DEAFNESS. 1. DEFINITION. 2. ELEVATION OF LOWER TONE LIMIT— A NEW CONCEPTION.

DR. MARK J. GOTTLIEB, New York.

PART I.

Previously, otosclerosis was applied to a form of deafness that progressively became worse. In addition to this, there was a raising of the lower tone limit, negative Rinné, prolonged bone conduction and a positive Gelle test. The symptoms from which a patient suffering with this form of deafness complains are tinnitus, paracusis, vertigo and loss of hearing. On autopsy in some of these cases, a fixation of the footplate of the stapes and a vascularization with resorption of the bone between the oval and round windows is found. Occasionally this vascularization associated with rarifaction (osteitis vasculosa, Alexander) is encountered in other parts of the labyrinthine capsule than the promontory. No definite changes have been found by careful workers in the membranous portion of the cochlea. Quite often, at autopsy of a case, that during life suffered with this type of hearing deficiency, no disease was observed either in the bony capsule or in the membranous portion of the labyrinth.

Furthermore, some of the essential diagnostic signs are not found in every case. This applies particularly to prolongation of the bone conduction. On testing the hearing from time to time, one is impressed with the fact that there is a variability in the results which could not be accounted for by a difference in the technic or the psychology of the patient. The Gelle test is not reliable because in cases where in life it was interpreted to be positive, in some at postmortem examination no fixation of the stapes was found. The only findings which are invariable and are always present are elevation of the lower tone limit and negative Rinné.

From the foregoing and on careful study of all the other information on the subject one must come to the conclusion that this form of deafness is not a distinct entity, but that it may with the addition of other facts be susceptible to subdivision into various groups which have similar symptomatology but on careful study are found to be different.

Kopetzky has added an important contribution to the findings in observing that the majority of cases of deafness of this type have a high or low calcium content of the blood and a high uric acid figure.

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This places such cases of deafness in the category of a "manifestation of a general disease".

We are not so sure that all cases of deafness of this kind are due to causes remote from the ear because it is quite possible that cases that have definite lesions in the bony capsule of the labyrinth may be essentially localized in the petrous portion of the temporal bone and the etiology may also arise in that vicinity.

We believe, however, that such cases that can be placed in the class described herein are due to constitutional causes. One additional fact strengthens that supposition. The author, in an article soon to be published in the Archives of Oto-Laryngology, has shown that in a group of cases of progressive deafness which he has examined

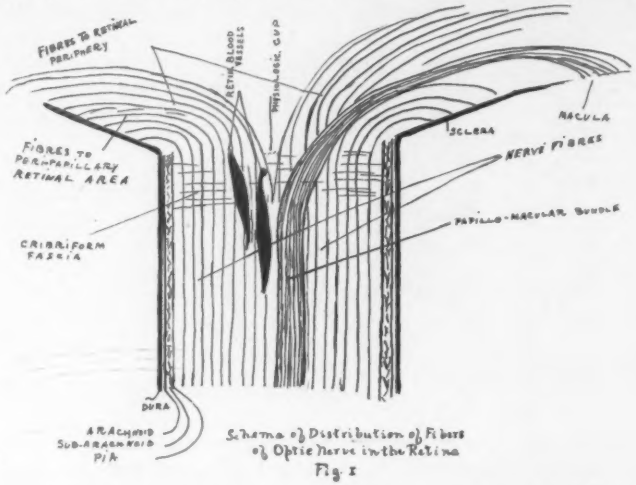

Fig. 1

recently, the visual fields are contracted for form and color. Since Dec. 9, 1927, we have had the opportunity of examining six cases of deafness which more or less conform in necessary detail to the signs and symptoms of the type of deafness under consideration. In all of these the fields were contracted for form and color, whereas normal cases and cases of other form of deafness showed no such finding. Now then, if this observation occurs with even less regularity in a large series of cases, it is reasonable to presume that the findings obtained on the examination of the eye may be used as a means of evaluating the character of the lesion in the cochlea and for this reason the author assumes the prerogative of naming the form of deafness in which all these signs and symptoms are encountered, "constitutional deafness".

In time perhaps with more careful study, cases of "constitutional deafness" may be subdivided into groups which may in turn be susceptible to the applications of different but rational therapy. Two cases were recently examined by me that suggest such a possibility. One was a case of psoriasis with deafness and obstructed Eustachian tubes, the blood calcium was low and the uric acid high. The visual fields were not contracted. The other was a case of deafness with a high calcium content in the blood, the visual fields were not contracted. For many years this patient has suffered with persistent looseness of the bowel movements.

To recapitulate—the diagnosis of constitutional deafness is postulated on the following findings: *1.* Elevation of the lower tone limit (constant). *2.* Negative Rinné (constant). *3.* Abnormal blood

Fig. 2.

chemistry figures (constant). *4.* Progression (constant). *5.* Contraction of the visual fields for form and color (variable?).

PART II.

We believe that the form of deafness described above occurs as the result of a disease process in the membranous portion of the cochlea, and particularly the cochlear nerve and its branches. It is therefore our duty to substantiate this theory by summoning the pertinent facts at our disposal. The most important evidence that it is a disease of the membranous portion of the cochlea and not the bony capsule is the occurrence of contractions of the visual fields in such cases.

Dr. Sigmund A. Agatston explains the occurrence of this finding as follows: "The result is suggestive of a relationship between the degenerate process of the ear apparatus and degeneration of the optic nerve as evidenced by the contraction of the visual fields in

these cases. Of course, we have not had a sufficient number from which to draw definite conclusions, but certain it is that visual fields must be taken in these cases and at some future date we may be able to make a more positive statement. Our findings correspond to a partial atrophy of the optic nerve, involving the central fibres of the nerve, though the papillomacular bundle seems unaffected. The reason that we localize the trouble in the center of the nerve is because in a general contraction of the peripheral visual field it is the center of the nerve that must be affected, since the central fibres are distributed radially to the periphery of the retina, while the peripheral fibres represent the area surrounding the disc as illustrated in the schematic drawing (Fig. 1). In the event that a distinct correlation is established, since this type of optic atrophy is generally

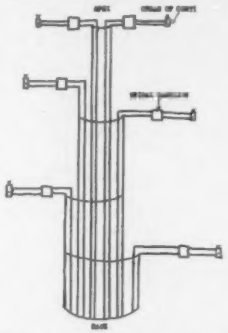

Fig. 3. Schematic drawing of arrangement of fibers of the cochlear nerve (Author's conception).

caused by some form of toxemia, one would infer from the eye findings that the disease of the cochlea is also caused by some form of toxemia. In none of our cases have I found atrophy sufficient to produce abnormal pallor of the disc. The appearance of the fundi was uniformly negative."

We know definitely that the fibres of the optic nerve that innervate the periphery of the retina assume a central position in the optic nerve. It is perfectly natural for the peripheral nerves to form the center of the nerve, otherwise they would have to reach the nerve trunk by crossing and twisting to reach a position other than the center. Nature works in direct and economical lines. It is more or less axiomatic that the longer a nerve fasciculus is the more centrally it is located in the nerve trunk.

There must be something in the involvement of the central fibres of the optic nerve that is analogous to the situation in the cochlea

that arouses our intense interest. It is the raising of the lower tone limit in cases of progressive deafness of constitutional origin. As has been stated before this is a constant and invariable finding in such cases.

It has been established that the high tones are perceived through the organ of Corti situated in the lower portion of the cochlea and the lower tones by that in the upper portion of the cochlea. In the case wherein the limit for the perception of lower tones is raised it is obvious that the nerve elements in the upper portion of the cochlea

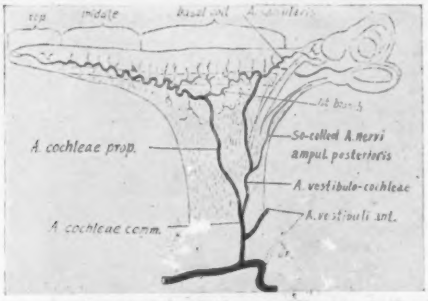

Fig. 4. Schema of artery in labyrinth, according to Author's observation (first kind).

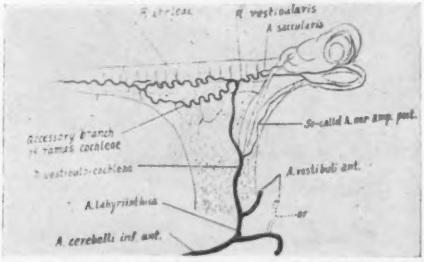

Fig. 5. Schema of artery in labyrinth, according to the Author's observation (second kind).

are impaired. Fig. 2 is a photomicrograph of the cochlea of the guinea pig cut through its center from apex to base, giving a good view of the cochlear nerve in the modiolus. It is the author's opinion that the nerve fibres from the upper coil of the cochlea occupy the center of the nerve trunk and those that enter the modiolus further down occupy a correspondingly more peripheral situation. This is schematically represented in Fig. 3. The portion of the cochlea which perceives the lower tones is comparable then to the periphery of the

visual fields. It is assumed by ophthalmologists that the central fibres of the optic nerve are particularly sensitive to the effect of certain toxins. No such idea is entertained relative to the fibres in the cochlear nerve which comes from the organ of Corti in the upper coil as compared to other parts of the nerve.

The circulation of the cochlea is such as to make the central fibres of the nerve more vulnerable. This is beautifully shown in the monumental work of Denjiro Nabeya on the circulation of the ear in man. He examined the circulation in 48 ears from 24 skulls and found that three types of circulation occurred in the cochlea of man. Figs. 4, 5 and 6 are taken from Nabeya's article and show the different types of circulation which he observed. These are schematically drawn with the cochlea straightened out. It will be noticed that

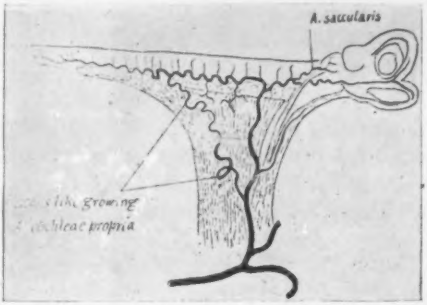

Fig. 6. Schema of artery in labyrinth, according to Author's observation (third kind).

the cochlea which has a circulation like that depicted in Fig. 4 is far more vulnerable than the other two types of circulation, because the collateral circulation ceases within the basal coil and beyond that the artery is a terminal vessel. This terminal vessel may be affected by poisonous substances with the production of hemorrhagic extravasations and exudates which may impair the nerves in the immediate vicinity and cut off the blood supply of the portions of the nerve and the hearing mechanism distal to it. This, in our minds, accounts for loss of ability to hear the lower tones even in the early cases of constitutional deafness.

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23 West 73rd Street.

BASILAR FRACTURE OF THE SKULL, WITH TEMPORAL BONE INVOLVEMENT.*

DR. WILLIAM G. SHEMELEY, JR., Philadelphia.

The purpose of the examination of any person with a head injury is to ascertain, if possible, the nature and extent of the injury and the procedure to be instituted that will best relieve and repair the resultant damage.

Fractures of the base of the skull frequently produce irritation or destruction of one or more of the cranial nerves. The seventh and eighth nerves are probably the most often injured. However, other symptoms produced are often manifested in the fundus of the eye or in other parts of the body.

Since this is true, then it would seem a rational procedure to have the otologist, ophthalmologist and neurologist examine the case, together with the general surgeon. The general surgeon usually regards, and rightly so, fractures of the base of the skull as more serious than fractures of the vault. The cerebral disturbance is more pronounced and vital parts are more often endangered in basilar skull fractures, since a greater degree of trauma is necessary to produce the fracture.

The fact that this type of fracture (basilar) frequently opens into cavities of the nasopharynx and ear results many times in septic infection as a complicating factor. Fracture of the base of the skull is often a concomitant of fractures of the vault. About 80 per cent of the fractures of the base of the skull originate as a linear fracture in the vault and extend to the base of the skull.

Where a fracture of the base of the skull has occurred, hemorrhage may take place from the ear, from the nose, from the mouth or may be subconjunctival. In a few cases blood will be noticed in all of these locations. A very small number of cases will present evidence of hemorrhage beneath the pharyngeal mucous membrane.

The writer has found, in the cases studied, that the order of frequency of injury to the cranial nerves in basilar skull fractures is first, the facial; second, the auditory; third, the sixth or abducens; fourth, the third or motor occuli; and fifth, the optic nerve.

Hemorrhage from the nose may be the result of fracture of the cribriform plate of the ethmoid. Should the olfactory nerves become involved in the fracture, impairment of the sense of smell may result.

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Fractures of the base of the skull more often involve the middle fossa. This injury, if the petrous portion of the temporal line is involved, often produces several signs of diagnostic importance: *1.* The escape of blood or cerebrospinal fluid, or both, from the external auditory canal or from the Eustachian tube. *2.* Deafness more or less complete and of sudden onset. *3.* Vertigo. *4.* Spontaneous nystagmus with the quick component directed away from the injured side, if the patient is conscious. If the patient is not conscious then there will be divergence of the eyes toward the injured side.

All or any combination of these symptoms may be present in a given case. Tearing of the tympanic membrane may result from the fracture. Should the bleeding be persistent it is indicative of fracture and is frequently mixed with cerebrospinal fluid. The possibility of this hemorrhage finding outlet through the Eustachian tube and being swallowed by the patient should be remembered. Cerebral tissue may appear in the external auditory canal or may escape from the nose. Infrequently blood may work its way forward through the sphenoidal fissure and the optic foramen and produce subconjunctival hemorrhage.

A fracture of the base of the skull that opens the pharyngeal mucous membrane is the result of very severe trauma. The hemorrhage is considerable. However, blood may be present beneath the pharyngeal mucous membrane without actual rupture. The mortality is high. In the cases studied, about 30 per cent of the fatal cases of fracture of the base of the skull died within six hours. About 60 per cent of the fatal cases of fracture of the base of the skull studied died within 12 hours. The death rate in this class of skull fractures will probably always remain high, since the very character of the injury places them in the class of primarily fatal cases.

Since the mortality will be high because of the very location of the injury, that should be an additional reason why the surgeon should avail himself of the assistance of the neurologist, ophthalmologist and otologist, that the proper procedure of treatment for the particular case under observation should be instituted quickly.

The case to be reported will prove of interest, the writer thinks, because there was involvement of the olfactory and auditory cranial nerves. In addition, there was damage to the optic, motor occuli, trigeminus, abducens, facial, gustatory and hypoglossus pharyngi cranial nerves on the left side.

The patient, A. J. V., male, age 48 years, reported for examination on May 26, 1926. Family history showed that his father was dead—

cause unknown. Mother dead—cause, sarcoma of the neck. There were eight children—one died in infancy, cause unknown. Two were stillborn in the early pregnancies. Four are living and well.

The personal history showed that the patient had had headaches almost continuous during waking moments for a number of years. He worked in a factory some years ago. The headaches have been less severe of late years. Slight attacks of rheumatism. On March 12, 1926, patient was injured in an automobile accident while motoring in California. Patient's head was pinned under car. He was not unconscious and he felt as if his left eye was protruding out of the socket. He was taken to a hospital where he remained for 16 days. No X-ray pictures were taken. Patient states he bled freely from the left ear, eye and nose. Left side of face, tongue and throat were paralyzed and still are, but not as bad as at first. He has very little taste and no sense of smell. Patient's left eye is turned in; can look up and down, but not to left—very little vision in left eye. Hearing in left ear was entirely gone, but has returned to some extent and is improving. Patient also had right shoulder fractured at time of accident. Sight in the right eye was impaired but has improved considerably—motion is all right.

Preliminary Examination—Nose: Fracture type deflection of the septum; dislocation of cartilage to the left; bilateral spine along suture line. Neither middle turbinate visible.

Teeth: All are in poor condition.

Throat: Examination reveals a secondary catarrhal pharyngitis. Tonsils are medium in size, deposits both sides on pressure.

Eyes:. O. D. moves in all directions. Test for convergence—right eye responds normally. O. S. motion inward, apparently normal, but no motion beyond midline. Motion up and down, normal. In taking movements of convergence, O. D. and O. S. respond equally. Ocular and palpebral conjunctiva congested, especially O. S. No secretion. Lacrimal apparatus, normal O. U. Cover test not made because of poor vision of O. S. Oblique illumination—cornea brilliant, transparent. Anterior chamber normal depth. Irides blue, except for several nevi; normal texture. Pupil round, black. O. D. 4 m.m. diameter, reacts promptly to light and accommodation. O. S.—pupil 7 m.m. diameter, reacts slightly to light but not to accommodation; however, when a strong beam of light is thrown into the left eye the right pupil contracts slowly. When a strong beam of light is thrown into the right eye the left pupil contracts promptly and equally with the right. Vision—O. D., 6/6. O. S.—fingers at 3 m.

Ophthalmoscopic Examination: Media clear O. U. Indirect O. D. —Disc elongated slightly; axis 70°. Well defined. Lamina cribosa discernible. Size and distribution of vessels normal. The fundus, including macular region, is normal. Direct examination—same findings as indirect.

Indirect O. S.—Disc fairly round. Well defined. The cup is poorly defined and filled in with connective tissue. Lamina cribosa not discernible. Bend of vessels not seen where they come over into cup. Size and distribution of vessels abnormal, in that the course of the vessels is slightly more tortuous and the vessels themselves are diminished in size. Macular region normal.

Right Ear: Conversational speech, 8 meters plus. Whispered speech, 8 meters. Acoumeter, 6 meters. Watch, 1.4 meters. C_1, short 80 seconds. C_4, normal. Air, short 20 seconds and short 25 seconds. Schwabach, short 5 seconds, short 5 seconds. Impossible to make Rinné.

Left Ear: Conversational speech, 6 meters. Whispered speech, 10 c.m. Acoumeter, 3 c.m. Watch, contact. Weber, referred to the left. Schwabach, short 5 seconds, short 5 seconds. Rinné, minus 25 seconds. C_1, not heard. C_4, short 8 seconds. Air, short 45 seconds, short 70 seconds.

With 2 m. speaking tube in the right ear and the noise producer in the left ear, patient hears whispered voice 8 inches from tube 100 per cent.

With 2 m. speaking tube in the left ear and the noise producer in the right ear, patient hears low voice 100 per cent.

Test for Spontaneous Nystagmus: When looking to the extreme right there is a mixed horizontal and rotary nystagmus to the right. When looking to the extreme left there is a slight amount of mixed horizontal and rotary nystagmus to the left. When looking straight ahead there is no spontaneous nystagmus. When looking up two or three vertical movements upwards.

Galvanic Labyrinthine and Eighth Nerve Test—Right Ear: Cathode 4 ma. produces a rotary nystagmus to the right. Anode 7 ma. produces a rotary nystagmus to the left.

Left Ear: Cathode 7½ ma. produces a rotary nystagmus to the left. Anode 12 ma. produces a rotary nystagmus to the right.

Turning Test: After 10 turns to the left, with the head erect, there is a nystagmus to the right lasting 25 seconds. After 10 turns to the right, there is a nystagmus to the left lasting 24 seconds. After

10 turns to the left, the horizontal nystagmus (afterturning) to the right was present in O. S. as well as O. D. After 10 turns to the right the afterturning nystagmus to the left was not present in O. S.

Otoscopic Examination—Right Ear: Membrane apparently intact, dull, opaque. Hammer handle foreshortened, retracted. Limited mobility with Siegel. Politzer inflation caused membrane to come out over small area and return promptly.

Left Ear: Membrane questionably intact, since there appears to be possible moisture in region of short process of hammer handle. Membrane is dull and opaque. Hammer handle foreshortened, denoting retraction. Limited mobility with Siegel massage. Politzer inflation and with good resistance, the membrane came out over limited area and returned slowly. No secretion in supposedly moist area.

Examination of Cranial Nerves: First, smell blunted (may be partly due to marked deformity of nasal bone resulting from the accident). Second, atrophy (*1.* Lamina cribosa not visible; *2.* blue-white pallor; *3.* cup filled in, do not see bend of vessels. Vessels more tortuous and diminished in size). Third, left is involved. Fourth, normal. Fifth, sensory below normal. Motor slightly subnormal. Sixth, involved. Unable to move left eye outward, beyond midline. Seventh, paresis of left; the right is normal. Eighth, abnormal. Vestibular and cochlear branches, dullness for hearing, not of pure nerve type. Ninth, slightly involved. Tenth and eleventh, normal. Twelfth, involved.

On May 26, 1926, the patient was referred to Dr. Weston D. Bayley for neurological examination, with the following result:

Examination revealed the left eye to be injured; pupil larger than right. There is a paresis of the left external rectus. There is almost complete seventh nerve paralysis on the left side; cannot close the eye. Hearing in left ear is impaired. Tongue cannot be projected fully and deflects slightly to the right. He has lost the sense of smell and the taste is much impaired. Taste is now developing on right side of tongue; absent on left. Sensation present in pharynx. The left pupil does not react to light; right is in the state of miosis; does not react, but there is movement in the color of the iris on throwing in a strong light. Knee jerks free; no ankle clonus. Some diminution of sensibility on left side. Patient states that condition has all cleared up in a measure. Fork on forehead and all over right side of skull only heard in left ear. No air conduction of same fork in left ear but can hear aerially a high pitch fork in left ear.

On May 28 the patient again reported to my office, when the following examination was made: *1.* Rhomberg negative. *2.* Gait for-

ward, eyes open, slightly hesitant but normal. *3.* Gait backward, eyes open, slight hesitancy but normal. *4.* Gait backward, eyes closed, hesitancy but normal. *5.* Gait forward, eyes closed, hesitancy but normal. *6.* Hopping forward, right foot, eyes open, normal; hopping forward, right foot, eyes closed, normal. *7.* Hopping forward, left foot, eyes open, normal; hopping forward, left foot, eyes closed, normal. *8.* Hopping back, right foot, eyes open, normal; hopping back, right foot, eyes closed, normal. *9.* Hopping back, left foot, eyes open, normal; hopping back, left foot, eyes closed, normal.

On June 4, 1926, *tuning fork test* was made, with the following result: Right ear: conversational speech, 8 m.; whispered speech, 6 m.; acoumeter, 5 m.; watch, 1.2 m. Weber to the left. Schwabach, short 25 seconds (bone referred to left). Rinné, +28″. C_1, short 14 seconds; C_4, short 3 seconds; air, short 30 seconds. Left ear: conversational speech, 6 m. and 3 m.; whispered speech, 1 m.; acoumeter, 1 m.; watch, contact. Schwabach, short 20 seconds. Rinné, —25″. C_1, not heard. C_4, short 13 seconds. Air, short 110 seconds. With noise producer in the right ear the patient heard conversational speech at 3 m.

Test for Spontaneous Nystagmus: When looking to the extreme right there is a mixed horizontal and rotary nystagmus to the right. When looking to the extreme left there is a mixed horizontal and rotary nystagmus to the left. When looking straight ahead there is no nystagmus.

Galvanic Labyrinthine and Eighth Nerve Test — Right Ear: Cathode 4½ ma. produces a rotary nystagmus to the right. Anode 6½ ma. produces a rotary nystagmus to the left.

Left Ear: Cathode 5 ma. produces a rotary nystagmus to the left. Anode 13 ma. produces a rotary nystagmus to the right.

Turning Test: Head erect, after 10 turns to the left there is a horizontal nystagmus to the left lasting 25 seconds. After 10 turns to the right there is a horizontal nystagmus lasting 24 seconds to the left.

Head forward 90°, after 10 turns to the left there is a rotary nystagmus to the right lasting 20 seconds. After 10 turns to the right there is a rotary nystagmus to the left lasting 17 seconds.

On June 11, 1926, the patient again reported. *Test for spontaneous nystagmus* made at this time revealed: *1.* When looking to the extreme right there is a mixed rotary and horizontal nystagmus to the left. *2.* When looking to the extreme left there is a mixed rotary

and horizontal nystagmus to the left, O. D. only. *3.* When looking straight ahead there is no spontaneous nystagmus.

Galvanic Labyrinthine and Eighth Nerve Test — Right Ear: Cathode 11 ma. produces a nystagmus to the right. Anode 5½ ma. produces a nystagmus to the left.

Left Ear: Cathode 11½ ma. produces a nystagmus to the left. Anode 13 ma. produces a nystagmus to the right.

Turning Test: Head erect, after 10 turns to the left there is a horizontal nystagmus to the right lasting 25 seconds. After 10 turns to the right there is a horizontal nystagmus to the left lasting 24 seconds. Head forward 90°, after 10 turns to the left there is a rotary nystagmus to the right lasting 16 seconds. After 10 turns to the right there is a rotary nystagmus to the left lasting 15 seconds.

On June 24, 1926, examination for *spontaneous nystagmus* revealed there was none when looking straight ahead.

Galvanic Labyrinthine and Eighth Nerve Test — Right Ear: Cathode 5 ma. produces a rotary nystagmus to the right. Anode 6 ma. produces a rotary nystagmus to the left.

Left Ear: Cathode 7 ma. produces a rotary nystagmus to the left. Anode 12 ma. no reaction.

July 21, 1926, *Galvanic Labyrinthine and Eighth Nerve Test— Right Ear:* Cathode 9 ma. produces a nystagmus to the right. Anode 11 ma. produces a nystagmus to the left.

Left Ear: Cathode 11 ma. produces a nystagmus to the left. Anode 15 ma. produces a nystagmus to the right.

Oct. 6, 1926, examination for *spontaneous nystagmus,* when looking to the right there is a nystagmus to the right. When looking to the left there is a nystagmus to the left. When looking straight ahead there is slight nystagmus to the left. $\frac{1 \times 1}{4}$

Galvanic Labyrinthine and Eighth Nerve Test — Right Ear: Cathode 8 ma. produces a nystagmus to the right. Anode 10 ma. produces a nystagmus to the left.

Left Ear: Cathode 10 ma. produces a nystagmus to the left. Anode 8 ma. produces a nystagmus to the right.

With 2 m. speaking tube in the right ear and noise producer in the left, patient hears conversational speech 100 per cent, even with mouth 4 to 6 inches from tube. With 2 m. speaking tube in the left ear and noise producer in the right, patient hears conversational voice 100 per cent correctly. Whispered voice, 60 per cent.

Oct. 19, 1926, *examination for spontaneous nystagmus,* no spontaneous nystagmus when looking straight ahead.

Galvanic Labyrinthine and Eighth Nerve Test — Right Ear: Cathode 9 ma. produces a nystagmus to the right. Anode 7½ ma. produces a nystagmus to the left.

Left Ear: Cathode 6 ma. produces a nystagmus to the left. Anode 11½ ma. produces a nystagmus to the right.

Turning Test: Head erect, 10 turns to the left produces a horizontal nystagmus to the right lasting 22 seconds. Ten turns to the right produces a horizontal nystagmus to the left lasting 20 seconds.

Head forward 90°, 10 turns to the left produces a rotary nystagmus to the right lasting 22 seconds. Ten turns to the right produces a rotary nystagmus to the left lasting 15 seconds.

Feb. 23, 1927, vision without glasses: O. D., 6/4½. O. S., 6/20 only with head to left side. Preliminary examination: O. D., eye moves well in all directions. O. S. moves with O. D. when looking to right, but when looking to the left beyond midline, motion is limited. Up and down, normal. Convergence fair. Ocular and palpebral conjunctiva congested. No secretion. Lacrimal apparatus, normal O. U. Cover test not made because of poor vision O. S. Cornea brilliant, transparent. Anterior chamber, normal depth. Irides, gray blue and except for several nevi, normal texture. Pupils round, black. O. D. 3 m.m. in diameter. O. S. 4 m.m. in diameter. Pupilary reflex to light active in O. D. Apparently absent in O. S.; however, when light is flashed in O. S., O. D. contracts; when flashed in O. D. there is no reaction in O. S. The pupils react to accommodation and convergence.

Ophthalmoscopic Examination: Media clear O. U. Direct O. S. Disc elongated. Axis 120°. Fairly well defined. Lamina cribosa unduly discernible. Color, white (atrophy). Artery and veins approach each other in size. Distribution of vessels normal. Fundus, including macular region, normal, except as noted.

Direct O. D., disc elongated. Axis 180°. Well defined; medium sized cup. Lamina cribosa distinctly discernible. Size and distribution of vessels normal. Fundus, including macular region, normal.

Retinoscopy: Right plus .87 Dsph combined with plus .25 Dcyl—180° axis. Left plus .75 Dsph combined with plus .25 Dcyl—90° axis.

Trial Case: Right plus .75 Dsph combined with plus .50 Dcyl—180° axis. Left plus 2.50 Dsph merely to fog as optic atrophy has damaged vision.

March 1, 1927, no muscle test made because of poor vision of O. S. Right plus .75 Dsph combined with .50 Dcyl—axis 180° equals 6/4½ vision. Left plus 2.50 Dsph (for cosmetic effect). Add for near R. plus 1.50 Dsph.

March 4, 1927, glasses neutralized and centered correctly.

Dec. 1, 1927, *tuning fork test, right ear:* conversational speech, 8 m. plus. Whispered speech, 6 m. plus. Acoumeter, 6 m. plus. Weber to the left, shortened 6 seconds. Schwabach, shortened 10 seconds. Rinné, +10 seconds. C_1, short 35 seconds. C_4, normal. Air, short 30 seconds.

Left Ear: Conversational speech, 1 m. Whispered speech, ad conch. Acoumeter, ad conch. Schwabach, shortened 10 seconds. Rinné, not made, no air conduction C_1, feels vibration but not sound. C_4, shortened 10 seconds. Air, feels vibration but does not hear. Weber was lengthened 6 seconds even when fork was 3 inches to right of midline of skull.

With Betzold A Forks patient hears with left ear equally as prompt as with right. The forks being moved to varying distances so that in each instance when he hears with left ear the right fork is far enough away that the sound of the left fork will drown out that of the right. With 2 m. speaking tube in left ear and Barney noise producer in right ear, patient hears low conversational speech 100 per cent correct. Whispered voice not heard. With 2 m. speaking tube in right ear and Barney noise producer in left ear patient hears both conversational and whispered voice 100 per cent correctly.

Otoscopic Examination, Right Ear: Impacted cerumen partially obstructs canal. After removing cerumen membrane found intact, dull and opaque. Short process of hammer handle unduly prominent. Long process foreshortened; slightly indirect. A moderate degree of retraction.

Left Ear: Cerumen, but not sufficient to obstruct details. Membrane apparently intact, dull and opaque. Short process of hammer handle unduly prominent. Long process slightly foreshortened, denoting retraction of moderate degree.

Examination for *spontaneous nystagmus:* When looking to the extreme right there is a nystagmus to the right. When looking to the extreme left there is a nystagmus to the left; equal to both sides. When looking straight ahead there is no spontaneous nystagmus.

Galvanic Labyrinthine and Eighth Nerve Test—Right Ear: Cathode 10 ma. produces a nystagmus to the right. Anode 11 ma. produces a nystagmus to the left.

Left Ear: Cathode 9 ma. produces a nystagmus to the left. Anode 13 ma. produces a nystagmus to the right.

Turning Test: Head erect, after 10 turns to the left there is a horizontal nystagmus to the left lasting 28 seconds.* After 10 turns to the right there is a horizontal nystagmus to the right lasting 30 seconds. Head forward, after 10 turns to the left there is a rotary nystagmus to the right lasting 22 seconds. After 10 turns to the right there is a rotary nystagmus to the left lasting 20 seconds.

January, 1928, in a telephone conversation with the Roentgenologist he stated that he had found a linear fracture of the base in the region of the petrous portion of the temporal bone and extending forward. Because of loss of some records the Roentgenologist is unable to give a more detailed report of his findings.

Laboratory Report: Urinary findings, negative. Blood and spinal Wassermanns, negative.

In concluding, the writer offers the following comments: *1.* Basilar fractures of the skull are of special interest to the otologist, since the seventh and eighth cranial nerves are the most often involved.

2. The death rate of basilar fractures of the skull will be high because of the nature of the injury and the parts involved.

3. Prompt co-operative teamwork between the surgeon, the neurologist, the ophthalmologist and the otologist offers a possible means of lessening somewhat the high mortality.

1724 Spruce Street.

*The first 10 seconds showed the excursions to be large and coarse, such as one frequently observes in cerebellar involvement.

MASTOID COMPLICATIONS: CASE REPORTS: (a) LATERAL SINUS THROMBOSIS AND CAVERNOUS SINUS THROMBOSIS FOLLOWING THE RADICAL OPERATION; (b) SUPPURATIVE LABYRINTHITIS AND MENINGITIS FOLLOWING THE SIMPLE OPERATION.*

DR. H. CLIFTON LUKE, New York City.

In submitting these two case reports the writer is mindful of the fact that these complications, *per se*, are not uncommon, but their frequent incidence as a complication of the particular operations in question is, I believe, unusual and of sufficient interest to report.

M. G., male, age 47 years, was admitted to Manhattan Eye, Ear, Nose and Throat Hospital, service of Dr. John R. Page, on Jan. 20, 1927, for a radical mastoid operation on the left ear. The patient had been under the writer's care in the clinic for about three months. He came very infrequently, and on the occasion of two examinations during this period the ear had appeared to be dry. The history dated back ten years, when an acute purulent inflammation developed on the left side following the use of a public swimming tank, the condition later becoming chronic.

On his admission the canal showed a thick, foul-smelling discharge. Only a small portion of drum in the upper segment remained. The malleus below the head had necrosed away and the internal tympanic wall showed a hypertrophic mucous membrane bathed in pus. Functional test of the ear showed complete inability to hear the whispered voice or acoumeter. The spoken voice was heard at 6 inches with noise apparatus in the other ear. The labyrinth appeared normally active with the rotation and cold caloric test. The right ear was normal.

A radical mastoid operation was performed on Jan. 20. Findings at operation were as follows: The antrum and vault of the middle ear were found packed with foul-smelling cholesteatoma, the inner plate over both of these cavities being eroded and necrotic. This plate was removed, exposing granulations on the dura, which appeared otherwise intact. Healthy dura was exposed on all sides.

*Read before the New York Acad. of Medicine, Section on Otology, Dec. 9, 1927.

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The mastoid tip, noncellular and sclerotic, was not removed. The sinus plate appeared normal, the sinus not exposed. The middle ear was thoroughly cleansed of granulations and bone debris and the Eustachian tube curetted. The wound was washed with Dakin's solution, 1:8, packed with plain gauze and closed with metal clips. The highest postoperative temperature was 100.2° on the following day, and practically normal thereafter to the tenth day, when the patient was discharged with the posterior wound healed, the cavity clean and general condition good.

He was admitted to the hospital Feb. 3 (14 days after operation) for secondary skin graft. The cavity was cleansed with normal saline solution and as the granulation tissue was not exurberant, curetting was considered unnecessary. Two grafts were removed from the thigh, with ice as a local anesthetic. One was placed in the tympanum and the other in the posterior part of the cavity extending to the external meatus. The patient's good condition and lack of elevation of temperature allowed him to return home the following day. He reported to the clinic daily. The packing was removed from the cavity on the fourth day and, except for a small area posteriorly, all of the graft appeared to be taking. On the fifth and sixth days the patient returned to the clinic for dressings and complained of headache on the left side. The next day (seventh since the grafting) the patient's son reported the former had walked the floor all night on account of headache. On examination shortly after, the man appeared acutely ill and presented a partial facial paralysis on the operated side, with temperature 100.2°, pulse 92. He was immediately removed to the hospital for observation. Following admission, the blood count showed a leucocytosis of 15,000, with 80 per cent polynuclear cells.

The next day a neurological examination by Dr. Zabriskie gave negative findings except for the peripheral facial paralysis, which was now quite complete and included the chorda tympani. All packing was removed from the cavity and boric acid irrigations given. During the following four days, to Feb. 18, the patient's condition remained practically unchanged. His temperature ranged between normal and 100.2°. Unilateral headache persisted but less severely. Examination for motor and sensory aphasia was negative. Eye grounds were normal.

On Feb. 18, thirteenth day after the skin grafting, the temperature reached 101.4° and it was thought best to curette lightly the cavity, which had almost completely filled with granulation tissue. This was done through the posterior wound under novocain anesthesia. Spinal

fluid taken at this time was clear and negative on culture. A negative spinal Wassermann was also reported.

During the next four days temperature rose gradually until it reached 104°. It was not of the typical sinus type, there being remissions of one or two degrees each day. Coincident with this the white cell counts had increased from 15,000, 85 per cent polynuclears, to 29,000, with 90 per cent polynuclears. Headaches had persisted, moderately severe, but there had been no chills, nausea or vomiting. Examinations had failed to establish any sensory or motor aphasia.

Blood culture had been negative until the afternoon of the eighteenth day, when a growth of nonhemolytic streptococcus was reported. The following morning examination of the spinal fluid was negative, but the eye grounds on the left side showed a choked disc. With a diagnosis of lateral sinus thrombosis the patient was immediately operated upon.

Operative findings were as follows: Early in the procedure a small amount of pus came from a sinus opening which pointed in the lower and forward part of the cavity. Deep pressure below and anterior to the tip of the mastoid forced 2 or 3 drachms of pus into the wound. The focus must have been situated internal to and in front of the mastoid tip near the stylomastoid foramen. There was no pain, surface tenderness, edema or infiltration to be observed in this region prior to operation. Provision was made to drain the pus pocket. The tip, which was noted as sclerotic and noncellular at the original operation, now appeared considerably softened. It was removed, as was also the plate over the lateral sinus from a point posterior to the knee above, down to the jugular bulb below. While the upper part of the lateral sinus was being exposed, the superior petrosal sinus was torn and bled freely until controlled. The lateral sinus was blocked off above and below and opened. A definite, partially occluding clot was found. The sinus flushed freely from above, but below, on removal of the gauze block, no bleeding occurred. The jugular vein was exposed in the neck and cut below between a double ligature. The same procedure was carried out above the common facial vein with ligature of the latter and removal of the resected portion of the jugular. The wound was lightly packed and left open. The external part of the sinus wall was removed along with the thrombotic mass. After the insertion of light gauze packing, the patient returned to the ward in fairly good condition. The blood had been previously typed and transfusion given.

The temperature ranged from 102° to 103° on the following day. The patient was dull and irresponsive and it was noted that a slight

exophthalmos on the operative side was present, which became more marked and bilateral as the day passed. He was irrational at times. The next morning the exophthalmos appeared more marked, indicating very definitely a cavernous sinus thrombosis. Death occurred the following day. No autopsy could be obtained.

This patient had an uneventful postoperative convalescence for 14 days, at which time the cavity was skin grafted. It was not until the fifth day following this (19 days after the original operation) that the first untoward symptom of headache developed. However, it does not seem probable that the skin graft was a causative factor in the complications. The collection of pus lying deeply near the exit of the facial nerve would readily explain the neuritis, with resultant peripheral facial paralysis. The mastoid tip was notably softened at the time of the sinus investigation, thus indicating the importance of the removal of this part when doing the radical operation. The bone appeared so definitely sclerotic that it had not been thought necessary. Infection from the pus cavity beneath the mastoid tip may have been transmitted to the sinus. It was at this point that the thrombosis was most marked. There is a possibility that the injury to the superior petrosal sinus may have been a factor in the cavernous sinus involvement, which was clinically noted within 24 hours after the sinus operation. It is, perhaps, more probable that this was brought about by way of the inferior petrosal sinus. However, an apparently fatal septicemia was in progress, so this feature might be considered to be more or less terminal.

A. S., male, age 9 years, came into Dr. Page's clinic at Manhattan Eye, Ear, Nose and Throat Hospital, April 21, with an acute purulent infection of the right ear which followed a nasopharyngitis three days before.

The patient was admitted to the hospital with a temperature of a little over 100°. He was discharged on the sixth day and the ear stopped running shortly after this.

About a month later, June 3, while the writer was off service, this boy was again admitted with a temperature of 102°, complaining of pain and discharge from the opposite ear (left). There was some edema and tenderness over the mastoid.

A simple mastoid was performed the following day by the house surgeon (under supervision) and aside from some exposure of the dura just above the knee of the sinus, nothing of any special interest is mentioned in the operative report. The temperature reached 103° the following day and the bedside notes state that the face and lips were red and swollen. He was put on erysipelas precautions. Swell-

ing persisted and on the second day red blotches appeared over the body, fading out at times. The temperature went to normal on the third day and with the disappearance of the rash and diminution of swelling about the face, precautions were discontinued. Temperature remained below 100° until the seventh day, when it again reached 101.° The following day at 6 p. m. it was 102.3°, with no special symptoms aside from very profuse discharge from both posterior wound and canal. During the next three days there was a return of the temperature to normal and the patient was then discharged.

One week later, June 23, the boy was brought to my office. He looked very pale and appeared acutely ill. There was active spontaneous nystagmus toward the normal side, *viz.*, to the right, a staggering gait, with apparently some tendency to fall to the right instead of to the left, as might be expected. The pulse was 140 and temperature 102.° With a noise machine in the normal ear he could not hear a loud shout, which suggested a dead cochlea. The mother stated that the child had been too ill to take to the clinic for the past two or three days as he had suffered much from headache, nausea and vomiting, dizziness and weakness.

The suggested diagnosis was acute diffuse suppurative labyrinthitis with probable beginning meningeal involvement. The case was immediately referred to the hospital. His temperature on admittance was 101.4°, reaching 104° the same evening. The blood count was 10,000, with 85 per cent polynuclears. A blood culture taken on the next day showed no growth after 24 hours.

Physical examination on the second day gave the following data: patellar reflexes not exaggerated, Babinski negative, Romberg sways to right, Kernig's sign very suggestive, spontaneous nystagmus to the right but less active. There was no evidence of any sensory or motor aphasia. Examination of the fundi showed no pathology. Passive movements of the neck gave moderate but definite discomfort. The patient complained chiefly of generalized headache.

Spinal fluid taken at this time appeared turbid and under moderate pressure. Report showed albumin 3+, globulin 3+, sugar negative, cell count 800. The complete failure to obtain any reduction in the Fehling's test was suggestive of meningitis. An X-ray of the head gave no additional information.

On the third day the temperature ranged between 102° and 104°. A report of the culture of the spinal fluid showed a growth of hemolytic streptococcus. Blood transfusion of 350 c.c. was given and later 1 c.c. of 1 per cent mercurochrome was injected into the spinal canal. Most of the day the boy appeared dull and irresponsive, complaining

of headache, but he would cry out occasionally and become very restless. Nystagmus to the right persisted but was less definite.

Without anesthesia the sinus was investigated and found to be normal. The dural exposure made at the time of the original operation was enlarged and meninges found markedly congested and under great tension. Three parallel incisions were made through the dura to relieve the tension. No evidence of injury to the horizontal or other semicircular canals could be made out. The patient's condition did not warrant any further examination.

During the night the patient became very restless and delirious. The neck was much more rigid. There were no localizing cerebral symptoms. Death occurred the following day, the final symptoms giving a definite picture of meningitis. Autopsy was refused.

It will be noted that this patient had a rather severe postoperative reaction, characterized by sharp temperature fluctuations, profuse purulent discharge and considerable cellulitis involving the face and postaural region, thus indicating an active organism with lowered resistance and presenting conditions favorable to extension of the infection to the labyrinth and meninges.

The question arises as to whether or not an immediate labyrinth operation, at the time of admission to the hospital, would have availed anything. It was believed then, however, that the meninges were involved, so this procedure was not carried out.

It is well known that labyrinthitis, following acute suppurative middle ear lesions, is more likely to be diffuse, with a meningeal involvement and end fatally than those developing in the presence of chronic suppuration with cholesteatoma. In the former there is no opportunity to build up any barrier of resistance and the host is overwhelmed. Such was the picture in this case, the meninges apparently being involved very early.

Hinsberg[1] states that a very large percentage of all cases of meningitis following middle ear disease are in reality secondary to an intermediate suppurative process involving the labyrinth.

121 East 60th Street.

1. HINSBERG, VICTOR: Ueber Labyrintheiterungen. Wiesbaden, 1901, p. 46.

FOLLICULAR DENTAL CYST. OPERATION.*

DR. HENRY M. GOODYEAR, Cincinnati.

Cystic tumors of the antrum have long aroused the interest of the general surgeon, dentist and, especially, the rhinologist.

Follicular cysts, with the body of the tooth, or teeth, in the lumen of the cyst and the root, or roots, embedded in the wall of the cyst (Fig. 1) are comparatively infrequent, hence such a case is recorded in this report.

The pathology of cystic tumors of the antrum have been very definitely and carefully worked out, and offer a distinct classification, namely:

Fig. 1. After Oppikofer.

1. *Ordinary Dental Cysts* (inflammatory cysts): Most common and arising secondary to an inflammation, producing a root granulomata. This granulomata by central absorption is converted into a cavity lined with pavement epithelium. Bruns traced embryologically the origin of the epithelium from the remains of epithelial rests of the embryonic pulp.

2. *Follicular Cysts:* Disturbance of development from abnormal tooth pulp (as reported in this paper). They develop from the tooth

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sacs and their genesis from the tooth roots is evident by the roots of isolated teeth in the cystic wall with the bodies of the teeth in the cystic cavity, as per Fig. 1 above.

Case Report: Mr. V. A., colored male, age 21 years, came on the Nose and Throat Service at the Cincinnati General Hospital, and was referred to my care by Dr. Iglauer on Nov. 5, 1927. There was considerable swelling and pain over the left antrum. The temperature was 104°, and the patient appeared to be acutely ill. A blood Wassermann was negative.

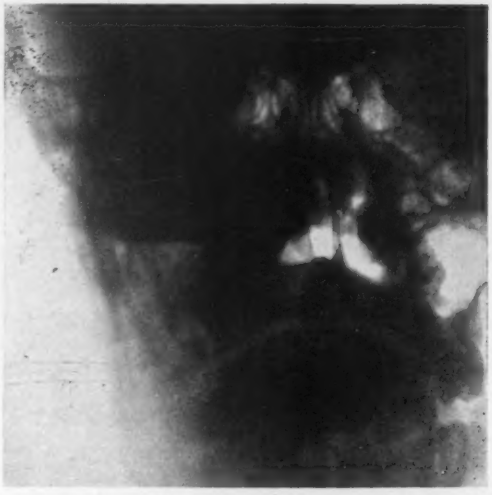

Fig. 2. No notation.

The swelling over the left antrum started 12 years before and had gradually enlarged, without pain, except during attacks of acute coryza. There was no history of profuse nasal discharge.

Upon palpation there was no "bony crackling"; the orbital floor was elevated and the roof of the mouth, left, was much depressed and the molar teeth completely covered by a firm swollen mass. The nose was clean and the eye findings normal. An antrum lavage returned only a small amount of straw-colored fluid.

The Roentgenograms revealed one tooth in the lacrymal angle and one well up in the lateral anterior wall, with the bodies of these

misplaced teeth pointing toward the center of the tumor mass (Fig. 2), thus suggesting a follicular type of dental cyst.

Operation: Five days after entrance the temperature had subsided and a radical removal was done. The walls of the antrum were very thin and a part of the maxillary bone was atrophic and came away with the tumor mass without cutting.

The tumor mass was very large (Fig. 3), and covered almost the entire body of my hand in outline. Fortunately the patient had a very large mouth, permitting the cyst to be removed *en masse*.

Fig. 3. No notation.

The pathological sections showed only fibrous and myxomatous tissues.

The healing was prompt after considerable bleeding during the first week.

I am indebted to Dr. Schell, of the Dental Department, for his assistance at the operation and for the blocking of the maxillary nerve by an external injection of novocain.

556 Doctors Building.

OSTEOMA OF THE FRONTAL SINUS— REPORT OF TWO CASES.*

Dr. Robin Harris, Memphis.

Gerber, in 1907, compiled from literature 87 true cases of osteoma of the frontal sinus, and since that date I find 28 cases reported; to which I wish to add two cases, making a total, through 1926, of 117. Osteoma in this region, although morphologically benign, is in a sense malignant. Growth into the brain may cause death by pressure necrosis, but more usually by secondary infection of the meninges, or the brain itself. Many good case reports are on record, the majority of which, prior to 1918, were presented by our European confreres.

Arnold, in 1873, made an excellent report of two cases of osteoma of the frontal sinus and his work seems more or less a connecting link between the old methods of surgery and our more modern ways of handling such cases.

Ardenne reported the case of a man, age 64 years, who noticed a polyp at the entrance of the left nasal fossa when 25 years old. Fifteen years later the patient noticed that he had near the inner angle of the eye a hard spot a little larger than a pea. This grew very slowly. At the of 58, the patient presented himself for treatment because of nasal obstruction from polypi and the growth at the inner angle of the orbit. Six years later the patient returned for consultation. Both nasal fossae were obstructed by polyps and the patient had severe pain over the frontal region. The tumor was removed, the frontal sinus walls were curetted thoroughly, ethmoid cells, polypoid masses and mucous membrane removed.

Probert gave a short description of a boy, age 15 years, who had a true frontal sinus osteoma. The only complaint by the patient was disfigurement by the enlargement at the inner side of the right eye.

Lang and Armour reported the case of a youth, age 19 years. In removing this tumor, Dr. Armour made a wide osteoplastic flap from above. This is not the usual incision, but was strongly recommended because in this case there was a large area of dural exposure.

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*Read before the Meeting of the Southern Section of the Amer. Laryngological, Rhinological and Otological Society, Memphis, Feb. 1, 1927.

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Goris removed a frontal sinus osteoma weighing 10 ounces. It is interesting to note that Goris for this operation did a tracheotomy and packed off the upper respiratory structures prior to attacking the growth.

Goodyear removed a large osteoma from the right frontal sinus under local anesthesia. The symptoms of terrific headaches were relieved.

Lee reported a case of osteoma of the right frontal sinus in which there was a history of trauma to this area by bumping the forehead against the end of a wagon about one year previously. In this case there was a tumor at the inner angle of the eye about the size of a small cherry. The globe was pushed forward, outward and downward, however, which would indicate that most of the enlargement was further back in the orbit. Only a part of the tumor was removed because of extension of the tumor in dangerous territory.

Sewall in one of the best of American articles on this subject reported a case in which headaches all of life was the predominating and practically only symptom. In removing this tumor Sewall used an incision through the brow. This was a small tumor weighing 47 grains and yet its removal relieved the patient of all symptoms.

Culbert in a masterly article described a case of an osteoma involving both frontals and the ethmoids. This patient had a positive Wassermann. The tumor was a very extensive affair, having eroded or absorbed much of the bone of the walls of the sinus, especially the posterior wall. A second operation was done, hoping to remove all the tumor, but the extension of this growth was so far back and involved so thoroughly the cribriform plate that some of it could not be removed. Three months later the patient died and was autopsied. There was much loss of tissue of both frontal lobes at the anterior and frontal surfaces. Multiple abscesses were found in both frontal lobes. Spicules of newly-formed bony tissue had penetrated the dura in many places.

Etiology: Arnold believed that osteomata arise from embryonic cartilaginous cells at the junction of the ethmoidal and frontal bones. In Case 2 of this report the origin was from the septum between the two frontal sinuses. Other observers believe these tumors arise from the periosteum of the sinus walls. Still others have advanced theories to the effect that they originate from the diploe. Chapman found in one case that the tumor arose by a short pedicle from the diploe of the skull above the frontal sinus. Pfieffer was of the opinion that osteoma were ossified polypi. Syphilis has often been mentioned as the cause, and in olden times mercury and iodids were

commonly prescribed for enlargements about the eye. During the past 20 years our laboratory methods have made the diagnosis of syphilis much more positive and we rarely now find syphilis given as a cause. Culbert's case is the only one in which I find record of a positive Wassermann. Trauma was given as the cause long ago and the history apparently was not complete until some recollection of trauma was obtained. As an example of this I will cite Cargill's case of a man who had had a hard blow on the face with a cap. On examining his face in the mirror this man noticed for the first time a facial asymmetry and apparent swelling of the right eye. He was then told by his friends that they had observed this condition for some months.

Boyd, in 1890, reported a case which had been observed for eight years, in which there was history of a kick on the forehead. One year after the latter, an enlargement was noted at the inner angle of one eye. The author states at the end of his paper: "I was not told about the discharge from the nose until after the operation or I should have concluded with some certainty that the growth sprang from the frontal sinus." The discharge was from both nostrils and continued after successful removal of the tumor. There was, therefore, undoubtedly an associated sinus disease.

Gerber, in 1907, made bold to state that sinus inflammation is the probable cause of osteoma in this region. Mr. Charles Wray, in 1915, strongly believed that sinus inflammation was the cause in all osteoma of the sinuses. He presented a specimen showing a bony excrescence which had not reached the stage of osteoma. Mr. Wray made the statement that there was only one specimen of genuine osteoma of the frontal sinus in the Museum of the Royal College of Surgeons. His observations are most interesting and because of these I am adding the report of a third case in this paper. Mr. Sampson Hanley removed from the frontal sinus a large tumor weighing 9 ounces, and among other things says: "It is interesting to note that the patient had suffered from bilateral nasal polypi for some years prior to the development of the osteoma." Axenfeld, in 1904, mentioned catarrh of the frontal sinus mucous membrane as a complication. He assumed, as many others do, that it was secondary to pressure by the tumor. In the description of his case, Chapman presented a clean-cut history of sinus disease; the patient, age 52 years, complained of headache of three years' duration. The headache began with an attack of grippe.

In obtaining the history of the patients it has been in the past the vogue to look for trauma, or syphilis, or tuberculosis, and evidence

of inflammation in the nose was overlooked. There is a class of people, rather a type of individual, who will minimize symptoms. Some refuse to put their mind on trivial deviations from the normal, especially if there be no pain, and others actually believe there is nothing wrong even when, for example, there is a discharge of pus from the nose all the time.

Attachment: Bessel-Hagen, in 1889, reported the attachment in his case to the junction of the frontal sinus and ethmoid bones far forward. Arnold apparently thought that this was the point of attachment in all such tumors. Birch-Hirschfield, in 1904, reported the attachment of the tumor, in the case that he operated, to the under and inner wall of the frontal bone and the ethmoid. Ardenne, in 1909, in his case found the pedicle relatively narrow and inseparable from its frontal and ethmoidal origins. Sampson Hanley found the attachment continuous with the frontal bone at the inner wall of the sinus and here its base was divided with a chisel. Goodyear found the origin in the upper lateral wall in the frontal sinus. Barnhill found his attachment at the infundibulum. In Hasting's specimen the attachment was to the floor of the frontal sinus external to, but not encroaching upon, the duct. The attachment in the first case reported below was at the anterior end of the junction of the ethmoidal with the frontal bone. In the second case the attachment was to the septum between the right and left frontal sinuses.

Growth and Size: The growth is usually slow. The fact that some of these tumors grow more rapidly following the operation is evidence that trauma may be an exciting cause or at least a cause for more rapid growth. As an example of this may be mentioned Thomas' case. In this patient only a part of the tumor was removed and 10 months later it was found to be larger than at the time of operation; one year later the tumor had increased considerably in size. In Case 1 reported below, we think this tumor increased markedly in size just a few months following the first operation. These growths range in size from very small and only discovered by X-ray, to the very large ones, as noted in the literature of 40 years ago. The tumor in Hanley's case weighed 10 ounces, Goris' 10 ounces. Some of the larger tumors come out in small pieces and it is impossible to tell how much they do weigh. Mention is made in Berg's thesis of a woman, age 64 years, who had an enlargement of the naso-orbital wall which had been growing for 22 years. It was so large that the cornea was displaced to a position only a little higher than the angle of the lips. This tumor dropped out spon-

taneously through a large ulcer in the skin. It weighed 10 ounces (Case of J. Irme, Centralblatt fur Angenhulkenndem, 1882).

Diagnosis: Before the days of the X-ray great stress was laid upon the enlargement, and up to the time of Berg great diagnostic value was placed upon the direction that the eye was displaced; if the eye pointed down and out the tumor was assumed to be of the frontal sinus; if directly out, of the ethmoid; if up and out, of the antrum. The X-ray is now our greatest aid in diagnosis. Recently two cases of apparent bony tumors of the frontal sinus have been discovered at the Memphis Eye, Ear, Nose and Throat Hospital. The Roentgenograms in these cases were made because sinus disease was suspected. The patient who is inclined to minimize symptoms or scoff at the possibility of his having any sinus disease will probably not be found to have a tumor until external enlargement takes place.

Symptoms: a. External deformity. This was formerly the only method of diagnosis. *b.* Headache is a symptom, usually an early symptom. In some cases the headache apparently begins with the development of the tumor. Sometimes headache is only a late symptom. Boardman and Donovan's first case was one of severe headache of long standing. The patient came with a diagnosis of brain tumor. The true diagnosis was made by skiagram. Sewall's case presented no external deformity and had complained of headache during whole life. *c.* Cerebral. Boardman and Donovan's second case complained of convulsive attacks. The tumor was found to be entirely within the frontal sinus. *d.* Discharge. History of discharge from the nose has been recorded in comparatively few instances. If a thorough search were made in each case it would probably be found that there is nasal discharge, or a history of recurrent discharge, in every case. *e.* Vertigo. Shea, of Memphis, has under observation two cases of apparent osteoma entirely within the frontal sinus, both of which were referred to him for Barany examination. Nothing was found by the latter test. The tumors were discovered by taking skiagrams for suspected sinus disease.

Birch-Hirschfield, in 1904, reported a case in which there was considerable doubt as to whether the tumor was an exostosis of the orbital wall or an osteoma involving the sinuses. In this case he used X-ray for diagnosis and found that by this method it was easy to differentiate between exostosis of the orbital wall and a tumor inside the sinus. In his case the tumor was easily seen in the sinus. He stated that this was the second time the X-ray had ever been used to assist in the diagnosis of such a condition. Sewall stated that Perthes was the first to publish the use of the X-ray for this purpose

but this was the same year that Birch-Hirschfield reported its use. Roentgenography has proved the deciding diagnostic factor in several cases. Among these might be mentioned the two cases reported by Boardman and Donovan, the one case by Sewall, and Case 2 of this report.

Case 1: This history is written as taken o nadmission. H. G., a veteran of the World's War, age 30 years, was admitted to U. S. Veterans Hospital No. 88, Sept. 13, 1926. He entered the hospital with a diagnosis of bronchitis, tachycardia, diabetis mellitus, and swelling above the left eye.

Fig. 1. (Case 1) Lateral Roentgengram before operation.

Father and mother both died of tuberculosis, one brother and one sister are living, one brother died of meningitis. The patient had never had typhoid fever, pneumonia, influenza, diphtheria or scarlet fever. He gave no history of being gassed during the war, and denied any venereal history. The patient first noticed a slight swelling over the inner angle of the left eye in 1923. He stated that this swelling had gradually enlarged, with no particular discomfort. The left eye was rather markedly displaced from its normal position: downwards 8 m.m., outwards 5 m.m., and forward approximately 2½ m.m. Examination of the eye did not show any pathology except possibly haziness of the left disc outline. There was no double

vision nor was there history of diplopia. The patient had only during the last few months had any headache and this not particularly remarkable.

General Appearance: The patient was a well developed and nourished white, ambulatory man, 67½ inches tall, and 125 pounds in weight. He presented a fair picture of health, stood erect, but there was a hard protrusion above the left eye, and this eye was displaced downward. The pupils reacted to light and accommodation. The nose was unobstructed. The tonsils were removed in 1924 and the throat was clear of inflammation. There was no inflammation or

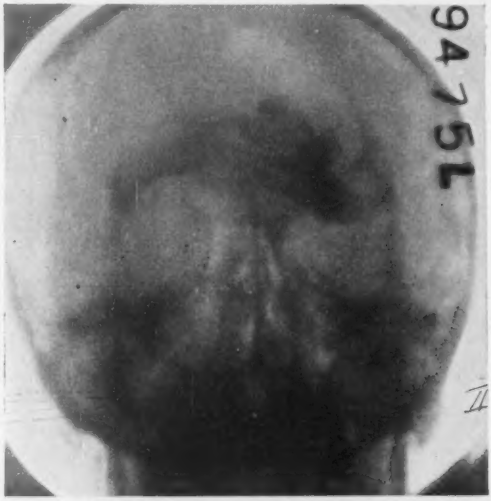

Fig. 2. (Case 1) Anterior-posterior Roentgenogram before operation.

discharge to be seen about the ears. Examination of the mouth revealed the teeth to be in filthy condition. There were present ill-fitting bridges, caries of several teeth and pyorrhea. There was no enlargement of the thyroid or cervical lymph glands. Heart sounds were normal and the blood pressure was 110-76. Examination of the lungs was entirely negative. Abdomen: There was a healed scar over the appendix region, but there were no masses or tenderness on deep palpation, no hernia, no varicocele. Motion of all joints of the body were free and there were no swellings. The Wassermann was found to be negative and there was a diabetis insipidus present but no diabetis mellitus. Examination by a neuropsychiatrist

indicated that the hysterical manifestations and tachycardia were due to irritation of the brain by the tumor.

Roentgenologist's report: "There is a very dense mass occupying practically the whole of the left frontal sinus, the left ethmoid region, and encroaching upon the left eye and the brain posterior to the frontal sinus."

Operation: Oct. 11, 1926, an incision was made through the entire left brow and 1 inch down the side of the nose. The incision was extended 1¼ inches upward near the center line of the forehead. The tumor, of very hard consistence, was noted presenting through the supraorbital margin about the center, extending upward about 10 or 12 m.m.; also the tumor was presenting in the orbit coming from the inner upper angle of the bony orbital wall. Practically all of the

Fig. 3 (Case 1) Before operation.

floor of the frontal sinus had been absorbed by pressure erosion; thin bony tissue was removed along the supraorbital margin (about 32 m.m. across and 35 m.m. upwards), giving a good view of the tumor, consisting of three stalks and a base; one of the stalks almost cauliflower-like in appearance, extended downward, outward and forward; at the largest portion it was perhaps 15 by 21 m.m.—this stalk presented in the orbit. The second portion was perhaps 30 m.m. long and 18 or 20 m.m. in diameter; this portion presented through the supraorbital margin. The third portion extended upward and backward, and was perhaps a little longer than the second portion. It had absorbed a portion of the posterior wall of the sinus, about 20 by 30 m.m.; that is, the dura was exposed in about that amount of space. These three pieces were attached, not very firmly, to a base below occupying the left superior plate of the ethmoid bone.

The three portions first described were covered with large polyps, the largest that I have ever seen. The three parts were easily removed. Escape of cerebrospinal fluid about the ethmoid made it advisable to pack the wound with iodiform gauze and wait. The entire operation was accomplished under local anesthesia. Twelve hundred milligram hours of radium were immediately given.

Thirty-five days after the operation the patient had four convulsions, tonic in type, each lasting about one minute. During these convulsions there was noted cyanosis, tossing of the body, much muscular contraction, and ejaculation of semen. After this the patient felt well, had no fever and no discomfort.

Fig. 4. (Case 1) Lateral Roentgenogram after operation, showing displacement of brain. A Cushing's dura clip is visible.

Seventy days after the operation the following report of eye examination was made by Dr. E. C. Ellett: "I saw H. G. and have the following report to make on his eyes: O. D. vision, 20/25, improved with —50 cylinder axis 90. The eyeball, eyeground and ocular movements and pupils are normal. O. S. vision, 20/20. The upper lid droops and the ball is on a lower level than the right, as if pushed down. Movement upward is defective, but no diplopia is presented nor can any be illicited with prism tests. The eye protrudes slightly, and as measured with the exophthalmometer there is only a difference of 1 m.m. in this respect. The eyegrounds and pupils are normal."

Second Operation: Jan. 11, 1927, local anesthesia. For this operation I secured the aid of Dr. Eustace Semmes, who limits his work to brain surgery. The head was shaved and the usual preparation made. Incision was 3½ inches straight upward, about ½ inch to the left of the median line of the forehead, and was extended outward along the brow at the site of the old incision nearly the whole length of the supraorbital margin. This triangular flap was elevated, and the opening in the anterior wall of the frontal sinus as noted in the outline of the first operation was found to be filled with polyps. The bone upward was very thin and appeared almost black. This was removed over the cavity shown better by the skiagram than by description. This cavity was filled with polyps, or rather a large polyp, which had pseudopods, extending, in at least two places,

Fig. 5. (Case 1) After operation.

through the dura. At one point this appeared to be connected with the parent polyp at the opening in the dura by an isthmus. The part inside the dura had to be coaxed, or, so to speak, milked out.

The brain was depressed backward perhaps 1½ to 1¾ inches. Below at the site of the ethmoid a large polyp and several small ones were attached to the hard bony growth which in front had grown upward along the anterior bony wall of the frontal sinus. This part was removed by biting with heavy forceps to a level with that part of the tumor occupying the left ethmoid region. A few strokes with mallet and chisel at the extreme anterior end loosened the base, which later was pried out. The base reached almost to the cellaturcica. It left its bed smooth, apparently complete. This entire area was then an opening into the nose. One silver clip was used in the dura and one point sutured with black silk. The incision was closed with no drainage externally.

There is no possible way to tell definitely just where this tumor was attached, but I believe the attachment was to the anterior wall of the frontal sinus at or just above the junction of the frontal and ethmoid bones.

The postoperative recovery is apparently complete. The patient has gained weight and strength rapidly. He weighs 20 pounds more than when admitted to the hospital.

Pathological Examination: Dr. J. A. McIntosh: "Specimen consisted of numerous irregular bony masses and larger yellowish-gray fibrous masses attached to some of the bony masses. On one surface

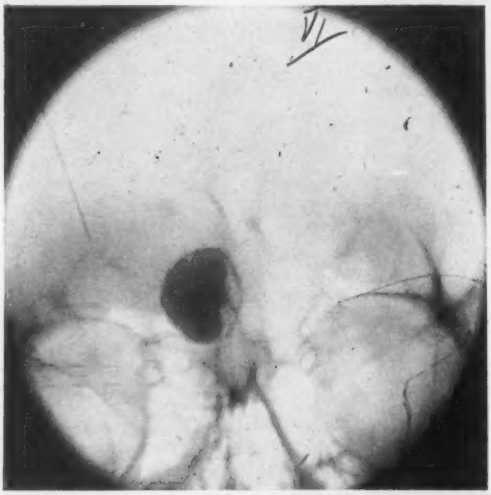

Fig. 6. (Case 2) Anterior-posterior Roentgenogram before operation.

the numerous pieces of bone were smooth and polished, harder than compact bone. The fibrous masses were largely globoid in shape, the largest 3 c.m. in diameter and on section presented a smooth, shiny, moist surface attached to the periosteum of the bony fragments. Microscopic examination of the soft tissue: One section showed a loose edematous, fibroid tissue covered by columnar epithelium, infiltrated by perivascular round cells and eosinphilic cells. There were numerous small petechial hemorrhages scattered throughout. The histology was that of a benign fibrous polyp showing congestion, edema and hemorrhage. *Diagnosis*: Chronic inflammatory fibrous hyperplasia with congestion, edema and multiple hemorrhages."

Case 2: A veteran of the World's War was admitted to the U. S. Veterans Hospital No. 88, April 19, 1926, for treatment of endarteritis obliterans of the right leg. The family history was unimportant. The patient had had measles, scarlet fever and attacks of tonsillitis. He denied venereal infection. The left leg was amputated three years ago for the same condition. His chief complaint was discharge from the right ear at intervals; the right foot was painful, cold and blue.

The patient was 69 inches in height, weighed 126 pounds, presenting a good picture of health. The right ear was discharging pus.

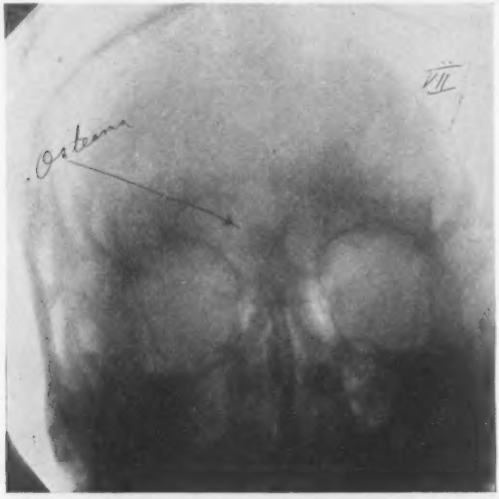

Fig. 7. (Case 3) Anterior-posterior Roentgenogram, showing a dense shadow in one frontal sinus and a general cloudiness of both frontal sinuses.

There was an apparent beginning atrophy of the mucous membrane of both sides of the nose. Throat: Posterior rhinoscopy showed the turbinates pale and pus was noted in the posterior ethmoid region on both sides. The tonsils had been removed. The hearing was: Whisper, 15 feet for the right ear, and 20 feet for the left ear. All teeth had been extracted and the examination of the chest and heart revealed no pathology. His blood pressure was 126-82. The pulse was full, bounding, regular and the rate was 100. No hernia, no varicocele, hemorrhoids or disease of the abdomen was noted. The right foot was cold and the great toe had a bluish tinge. The Was-

sermann proved to be negative. X-ray of the sinuses revealed much increase in density in all the sinuses on the right side, and there was present a very dense kidney-shaped body within the right frontal sinus. The Roentgenologist's diagnosis was bone tumor of the right frontal sinus.

Operation was done under local anesthesia, March 28, 1926. Incision was through the brow and ¾ inch down the side of the nose. The floor of the frontal sinus was removed and the sinus found to contain a hard marble-like body, thickened mucosa, a small amount of mucopus, the latter especially at the entrance of the natural opening and far externally. The incision was extended upward near the median line about ¾ inch and a part of the anterior wall of the frontal sinus removed. A hard bony tumor was then pried loose from a sessile attachment to the intrafrontal septum a little below the middle of a line drawn from the top to the bottom of the frontal sinus. The removal left a small opening in the very thin septum. The tumor was removed without marring or cutting. The frontal sinus was cleansed of mucous membrane. The ethmoid cells, showing rather marked inflammation, were removed and drainage made into the nose as for a Lynch operation. The evidence was that this sinusitis was of long standing and it may have been the cause of the recurrent attacks of purulent otitis media. The tumor in no way came in contact with the opening of the frontal sinus into the nose. It may be possible that this sinus inflammation was an exciting cause for the osteoma, but the latter could not have been the cause of the sinusitis, in that no blockage was possible. Recovery from the operation was rapid and uneventful.

Pathological Report of Tumor and Mucous Membrane: "Clinical diagnosis: Osteoma of the right frontal sinus. There are a few strips of soft tissue resembling mucous membrane and one bony tumor growth. This eburnated body tumor contains two faceted areas. There is a definite pedicle 6 by 6 m.m., by which the mass was attached to the adjacent bone. The mass has a grayish appearance and is stony hard. Diagnosis: Osteoma of the right frontal sinus." (Hamilton.)

Case 3: J. A. P., a veteran of the recent war, age 30 years, entered U. S. Veterans Hospital No. 88, Oct. 8, 1926, for examination and observation because of attacks of asthma.

The patient was a member of a family of 16 children. He had had most of the contagious diseases of childhood, and in service he had chills and fever. He stated that he was gassed and sent to a hospital in France for treatment of same. The present illness began in April,

1920, with an attack of asthma and he stated that the trouble with his stomach began at the same time. The chief complaint was difficulty in breathing, gas on stomach and headache.

The examination revealed the physical signs of bronchial asthma, enlargement of the prostate and chronic pansinusitis on the right. There was much pus in the right side of the nose, and the skiagram showed marked increased density in all the sinuses on the right side, together with a very dense area in the right frontal sinus. The appearance of the latter was that of a bony tumor. The Wassermann was repeatedly negative. Provocative Wassermann was also negative. The prostate was treated and improved much during hospitalization. The sinus condition did not improve. Headaches continued, as did discharge of pus from the nose. The patient refused operative interference, but he was kept for more than a month afterwards because it was the opinion of the hospital staff that the sinus disease was responsible for his bronchial asthma. Finally after repeated efforts to get the patient to undergo a sinus operation, he signed out against medical advice and no more has been heard of him.

This third case is presented because the bony growth in the frontal sinus was small, and because there was a marked chronic inflammation of the frontal sinus. The small dense body within this frontal may be a beginning osteoma and the study of such cases may lead us to gain in the future positive information as to the cause of osteoma of the sinuses.

The first 28 cases are operations since Gerber's tabulation 20 years ago.

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A RARE OR HITHERTO UNRECORDED CAVITY WITH FLUID IN THE BONY NASAL SEPTUM AND THE CRISTA GALLI.*

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I. The privilege of presenting the description of a condition of which I cannot find another example in the literature, comes to me at this time. I ask the indulgence of the Academy for writing in the first person throughout, but the nature of the report is such that some of it could not well be written otherwise.

The subject of cavity in the septum and crista is of threefold importance: first, clinically, because through distention of the septum there appear symptoms of nasal obstruction (between the septum and the middle turbinates); second, surgically, since, as will be shown later, recognition of the condition at operation is a highly desirable matter; and third, anatomically, because of the somewhat unusual method of development of these cavities, and because of their astonishing (yet heretofore unsuspected) frequency, especially in the crista.

A long and painstaking search of the indexed literature revealed no instances of septal cavity with fluid, nor any of cavity in *both* septum and crista, with or without fluid. Of a great number of old and new textbooks examined, both English and German, not one could be found to mention the subject; this includes Körner, Denker-Brünings, Grünwald, Thomson, Coolidge, Gleason, Kyle, Loeb, McKenzie, Packard, Dunning, Skillern, Reik, Ballenger, Ingals, Knight, Bosworth, Sajous, Coakley, MacDonald, Wright, Logan Turner, Porter and Ball. Bishop showed an illustration wherein the septum seemed to have two plates, apparently fused at the top and bottom; no adequate explanation accompanied this. Pratt's volume on intranasal surgery shows several excellent examples of pneumatized crista galli, and calls attention to these. Burnett illustrated a specimen in which two septal plates were present in the superior portions, and he attributed this anomaly to imperfect fusion of the perpendicular plate—an impossible explanation. The older Shurly went into a detailed discussion of Wright and McDonald's ideas regarding the pathogenesis of cystic turbinates, but did not mention septal cavities.

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Gruber, in 1875, described a cavity in a crista galli. Sercer, in 1926, discussed a septum which was doubled in its anterior portion. F. J. Pratt has encountered, but never reported, two cases of moderate-sized cavity in the bony septum, apparently not involving the crista, each of which contained a little clear fluid. Hallinger described, last July, a large cavity or cell in the perpendicular plate of the ethmoid, its roof being "a part of the cribriform plate". This, therefore, did not extend into the crista and it certainly did not contain fluid. McCullagh, in 1910, reported a "bone cyst" of the septum—a large, dry cavity in the vertical plate, with very thin walls. However, true bone cysts of the septum, while not impossible, are practically unheard of, so that it is one thing to call a given cavity a cyst and another to prove it. Microscopic study of a piece of the lining membrane is essential to such proof, and such study was not made in McCullagh's case (or if made, was not recorded in the report). Neither was such study made in my own case, for I did not know then what I know now about such cases, and it is for that reason that I call the condition simply a cavity, so that it may have been either a cell or a cyst. Furthermore, I appreciate fully the fact that my inability to find a report of such a case does not mean that no such report has ever been made. The title of this paper has been worded with that possibility in mind, although, as already stated, a long and fruitless search over the periodical and textbook literature of many years has not revealed any similar case.

II. *The Case.* On Nov. 8, 1926, Mr. E. B., age 20 years, consulted me for the relief of severe nasal obstruction, of several years' duration. Examination revealed a septum which was very thick between the middle turbinates, moderately deviated, and with a rather wide spur to the left inferiorly. A submucous resection was clearly indicated. On Nov. 10, the operation was begun in the usual manner; the mucous membrane, together with the perichondrium and periosteum of each side, had been elevated, and the necessary portion of septal cartilage had been removed. The bony septum appeared to be very thick. Bone forceps were inserted just above the lower edge of the perpendicular plate, the jaws were closed and I was surprised to find very little resistance to the closure. I withdrew the forceps, glanced at the fragment removed, then reinserted the forceps preparatory to continuing the procedure, when I was astonished to find a considerable quantity of fluid flowing down from the bony wound. The septum was hollowed out, and a thick, viscid, greenish-gray fluid trickled from the cavity. I felt certain that this could not possibly be a meningocele or any other sac with an intracranial connection because the secretion did not in the least

resemble cerebrospinal fluid. For this reason, I concluded that I must here be dealing with a cyst, although I had never heard of a cyst in this location. Accordingly, I proceeded very gently to probe the walls of the cavity, having first carefully ascertained the level of the cribriform plate at each side of the septum. To my surprise I found that there was no bony vault or roof in the cavity at the level of the cribriform plate, or even a little above it. I showed the case to F. J. Pratt, who looked into the cavity and also probed, a little higher than I had done, with the result that we finally found a bony roof at about the level of the top of the crista galli.

The cavity was about ½-inch long (anteroposteriorly), from ⅛ to ¼-inch wide, and reached upward to a level more than 1 c.m. above the cribriform plate. The walls seemed smooth, but the lumen appeared a little constricted at the cribriform level, and I did not probe the side walls above this level, though it would have been extremely interesting to find an opening from this cavity into an adjoining one. As we shall see later, the bony vault encountered here may have been the top of the crista galli or, more anteriorly, the roof of a frontal sinus. The flow of secretion stopped after a few minutes; from 3 to 6 c.c. of fluid had escaped. The operation was then continued, removing the two walls of the cavity as is usually done with the perpendicular plate. The postoperative result was good: a straight, thin septum, without subsequent distention and with adequate breathing space on each side. When several months later it was decided to report the case, the patient had moved to a different state. Roentgenograms of the nose and sinuses had not been taken prior to the operation, though some have been taken since.

III. *Embryologic Considerations.* You will recall that in the early embryo four pairs of branchial arches are found. The first arch on each side bifurcates early, forming maxillary and mandibular processes, the latter soon uniting to form the lower jaw. The second arch becomes the hyoid, and the third and fourth sink into the cervical sinus and become obliterated.

The frontal process of the head is divided early on each side by the olfactory pits to form lateral and medial nasal processes. Then the maxillary process mentioned above unites first with the medial nasal process of each side to form the upper jaw, then with the lateral nasal process to form the cheeks, parts of the lip, and the alae nasi. Later the two medial nasal processes unite to form the medial part of the upper lip. At the same time the medial remainder of the original frontal process of the head becomes compressed to form the thick, primary, membranous septum and the dorsum nasi.

The olfactory pits deepen inward, approaching the pharyngeal cavity posteriorly, finally breaking down the oronasal membrane and thus forming the primitive choanae. At the same time the primary septum, which at first was an anterior, or ventral structure, grows posteriorly or dorsally toward the pharynx, and downward or caudally toward the mouth. The latter is not as yet closed off from the nasal cavity because the hard palate has not fused across. The extension of the septum above mentioned is the secondary or definitive septum; it develops a free border posteriorly, dividing the choanae, and finally joins the fused hard palate below it to separate the nasal from the oral cavity in this region. This secondary septum is relatively thin, and the entire septum now becomes gradually thinner. A laminar plate of cartilage develops medially in its substance. This medial situation is important to us, for while a part of this cartilage persists later as the quadrilateral and the vomerine cartilages, other parts (in the secondary septum) are replaced by bone to form the perpendicular plate and crista galli of the ethmoid. In other words, these latter ossify as *median structures, and for this reason cavities within them cannot be explained upon the basis of imperfect fusion.* The vomer, on the contrary, develops bilaterally in the inferior and posterior part of the septum from an ossification center on each side of the cartilaginous septum, beneath the border of which they unite and grow upward as a bony plate on each side of the septum, enclosing the cartilage between them. These plates unite from behind forward, compressing or absorbing or pushing the cartilage ahead of them, until at about 15 years the union is complete. In the adult we see only a groove between these two alae, and in this groove, anteriorly, the septal cartilage reposes. It is not uncommon to find, as a result of imperfect or incomplete fusion of the two vomer plates, a fairly definite cavity, often of considerable size.

The paranasal sinuses are primarily evaginations of the nasal mucosa from beneath the middle, superior or supreme turbinates. We must not lose sight of the fact that the activity is in the epithelial tissue of these advancing projections. Coupled with this there is absorption of the bone, so that as a result of the wandering of these sacs into the ethmoid, frontal, maxillary and sphenoid bones, corresponding sinuses are formed. The point where a given sinus first leaves the nasal fossa is its permanent ostium. Conchal or turbinate cells are only outlying ethmoid cells and develop in the same way.

IV. *Anatomy of the Bony and Cartilaginous Septum.* This subject is so familiar to rhinologists that I shall only mention the various structures which enter into the formation of the septum. The

vomer posteroinferiorly and the perpendicular plate of the ethmoid superiorly comprise almost the entire bony septum. In addition we find anteriorly the frontal spine of the frontal and the crest of the nasal bones; posteriorly the crest of the sphenoid with its rostrum below; and inferiorly the crests of the maxillary in front and the palate behind.

The most important of the septal cartilages is, of course, the so-called quadrilateral or triangular cartilage. It may be well to recall that occasionally it gives rise to a posterior prolongation, extending between the vomer and the perpendicular plate of the ethmoid, and called the "sphenoidal process of the septal cartilage". This is often the cause of spurs or other deformities in this region. The vomero-nasal cartilages frequently cannot be distinguished from the septal cartilage, being often fused with it along its inferior border. They are two long, narrow strips which lie between the septal cartilage above and the maxilla and vomer below. The medial crura of the major alar cartilages form a part of the most anterior portion of the septum.

From this point on, in speaking of "septum" I shall refer constantly to the bony septum unless otherwise specified.

It should be noted that the perpendicular plate of the ethmoid is larger than generally supposed; it descends to the level of the lower border of the middle turbinate or the upper border of the inferior.[5] It is therefore evident that if pneumatization of such a bone should set in, the cell could attain large proportions without having to invade any other bone (although such an invasion would be easily possible). The degree of nasal obstruction resulting from this condition would be great. I have no doubt that this very situation is present in many persons at this moment, and it is well to be prepared to recognize the probability of a cavity in such a septum upon examination.

V. The parana al sinuses can assume innumerable variations of form. Not only this, but they can invade and pneumatize many adjacent struc ures in a surprising manner. For instance, the turbinates are very frequently found to contain a cell, as mentioned above.

About two months ago while meditating on this subject and studying a section of a skull specimen which was prepared for me some time ago, I suddenly noticed that the right frontal sinus had unmistakably begun to invade the crista galli posteriorly and was trying, apparently, to push downward into the substance of the septum. Such a possibility had not occurred to me before, yet here was unquestionably a definite pneumatization of the crista by the frontal sinus, whether this sinus had any designs upon the septum or not.

This gave me an idea as to a possible etiology, so I wrote to J. Parsons Schaeffer, whose authority in the field of rhinologic anatomy is undisputed. Dr. Schaeffer stated that he had never seen a bone cyst of the vertical plate of the ethmoid, though it may occur. He had, however, seen a number of specimens in which a recess expansion of one of the frontal sinuses had extended dorsally and invaded the crista galli, while in a few of these cases the pneumatization had extended well down into the perpendicular plate, causing a marked widening of the septum in this position. Such a recess would therefore communicate with the frontal sinus and so indirectly with the middle nasal meatus; the lining membrane would be similar to that of the frontal sinus. The very dependent situation of such a diverticulum of the frontal sinus would account for the collection of fluids in the bottom of the cavity. Dr. Schaeffer added also that he has seen specimens wherein a sphenoid sinus had invaded and pneumatized the septum from above and behind, causing marked widening of the septum in this region.

The idea of regarding these cavities in the crista and the vertical plate as simple outpouchings of the frontal sinus is a matter of great importance, because the presence of the fluid found in my case is thereby easily explained. If, for instance, the patient had had a low grade sinusitis and the customary secretion had formed, it would remain indefinitely in the septal cavity through lack of an outlet. The only possible exception to the idea of frontal sinus origin of these cavities might be that a number of X-ray pictures of young children's sinuses reveal an apparently pneumatic crista *without* a well developed frontal sinus. However, this pneumatization is usually not real, only apparent, since in young children the cancellous bone in the center of the crista is so soft and loose that it scarcely throws a shadow on the plate. In the few real cases, a frontoethmoidal cell or a high ethmoidal cell is generally found which can easily expand into the crista galli and pneumatize it.

I have studied the matter by means of X-ray plates as ordinarily taken for the diagnosis of sinus conditions. These plates are not always satisfactory because of the superimposition of other bony structures, because of the variations in bony density, and because of the difference in distance of various structures from the X-ray plate. For the most part, however, they are very instructive, and much can be learned from them.

Out of a group of more than 4,000 X-ray plates of all kinds, most of which were taken at the Minneapolis General Hospital, a series of 807 sinus plates were selected for study. Plates of infants and of very young children were not included.

Fig. 1. Unusual case showing the communication of a septal cavity (a) with a frontal sinus (y) (retouched).

Fig. 2. A septal cavity (a) communicating with a frontal sinus (y); the crista galli (x) is pneumatized (retouched).

Fig. 3. A frontal sinus (y) invading the septum (a); pneumatic crista galli (x) (retouched).

Fig. 4. Cavity in crista (x); all sinuses on both sides are cloudy except the right frontal (y) and ethmoids (z), showing a definite connection between these and the clear crista.

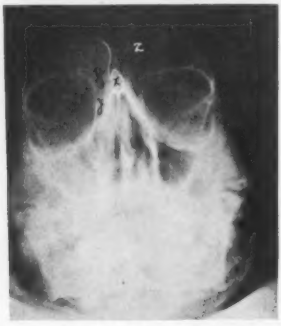

Fig. 5. Pansinusitis of one side, with involvement of the crista (x) which must therefore have been pneumatized from this side (y), since the opposite side (z) is clear.

Fig. 6. A large sphenoidal cavity in the septum (b) (retouched).

These pictures were divided into four general classes: Class I: 518, or 64 per cent, showed a solid bony crista and septum. We are accustomed to think of all septa and cristae as solid, hard bone; this is true, however, of only about two-thirds of all cases. Class II: 142, or almost 18 per cent, displayed a crista with a firm, cortical bony shell, but with a center of loose cancellous bone. This condition is by far the more common in children, being here the rule, rather than the exception. As a corrollary we might say, therefore, that the great majority of cases in Class II was composed of children under 13 or 14. Class III: 136, or 17 per cent, showed a pneumatized, more or less distended crista galli. This is an astonishing figure, since it means that one crista out of every six or seven is at least partly pneumatized. Class IV: 19, or more than 2 per cent, presented a definite cavity in some part of the bony septum, with or without a pneumatized crista. Some of these septa were pneumatized from the frontal sinuses above, others apparently from the sphenoids behind. Unfortunately, it is rarely possible to show in a Roentgenogram the exact point of communication of a septal or cristal offshoot with the parent sphenoid or frontal sinus. The nature of its development, together with the presence of many confusing pockets, trabeculations and superimposed structures in this area, render a sharp picture of such a communication almost impossible to obtain. Rarely, however, such a picture may be found.

An interesting item in the plates of children of from 2 to 12 years of age is the great clarity and constancy with which the two bony plates of the vomer, previously mentioned, were seen to surround the lower part of the original septal cartilage. This is most prominent before the age of puberty, but can be occasionally made out in the plates of adults.

The plates were all taken in the chin-nose position, which best shows the frontal sinuses and adjacent structures, including the upper part of the septum. While the plate is taken, therefore, the frontal plane of the head is not parallel with the X-ray table, that is, it is not exactly horizontal; the chin end of the head is lower than the forehead end. Frequently the entire dorsum nasi, because of this position, assumes a plane almost directly perpendicular to the X-ray table, and for that reason produces a heavy, clear shadow. In such cases the crista galli may seem to lie partly above and partly below this nasal roof, and it is well to remember that this is only a matter of position during the exposure. The pictures shown on these plates are enlarged about 25 per cent as compared with the original subjects, therefore a little allowance must be made for size in the structures seen.

Attention must also be called to a few other items. Frequently a long, median streak will be seen to extend upward from the vicinity of the crista galli. This is the shadow of the crista frontalis, a well-marked thickening on the inner aspect of the frontal bone. The shadows of these two structures are, of course, entirely independent of each other, although they are often superimposed, so as to give the crista galli the appearance of being prolonged far upward. We must be on the lookout for a septum which is so sinuous and tortuous that, by seeing different parts of the septum at varying distances from the plate, we get the erroneous impression of a split septum with a cavity. Usually we can notice a difference in the sharpness and intensity of the two linear shadows, so that one of these must be in focus while the other, being out of focus, is nearer to or farther from the plate than the first. Very frequently it is a very difficult matter to decide whether a given shadow denotes a cavity or a center of cancellous bone. The difference in density must guide us: if the appearance is like that of the frontal sinus itself or the orbit, we are dealing with a cavity, if otherwise, with loose bone, unless we should happen to find another cavity containing thick fluid, in which case this would probably cast a shadow not much different from that of cancellous bone. We must also note the important fact that a cavity in the septum which is closed *above* as well as below is probably a forward outgrowth of one or both sphenoid sinuses into the septum. Occasionally the parallel walls of this cavity can be seen posteriorly, with the undivided central septum superimposed anteriorly.

In the foregoing I have tried to show that this subject has very definite anatomical, clinical and surgical significance. If I shall have succeeded in accomplishing this purpose in any appreciable degree, I shall consider myself amply rewarded.

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507 Physicians and Surgeons Building.

SEQUESTRUM OF THE SUPERIOR MAXILLA FOLLOWING TYPHOID FEVER.

DR. G. M. VAN POOLE, Honolulu, T. H.

Mrs. S. I. entered the Japanese Hospital, Honolulu, on Feb. 1, 1927, suffering from a fever, which was afterwards diagnosed as typhoid. She remained in the hospital until May 28, 1927, when she was discharged from that institution. On June 30, 1927, she re-

Fig. 1.

Fig. 2.

ported to us on account of the right side of her mouth being sore. She complained of pain every time she tried to eat and said it was difficult for her to talk. Upon examination it was found that she had a sequestrum of practically the right half of the superior maxilla. By grasping the teeth and moving them with force a portion of the right superior maxilla would move, including the alveolar process, as far forward as the canine fossa, the palate process complete, and a portion of the body. The line of division between healthy and

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necrosed bone ran up from between the bicuspids along the canine fossa and thence backwards, including a portion of the malar process (see Figs. 1 and 2). Wassermann was negative, but she had a positive Widal. On July 15, 1927, under local anesthesia, the sequestrum was removed along the line of cleavage, as stated above. This left an opening into the antrum the size of one-half the mouth (see Fig. 3). From the antrum two slit-like openings, the lower about 3 c.m., and upper about 2 c.m. in length, allowed communication between the antrum and the nasal fossa. After the removal of

Fig. 3.

Fig. 4.

the sequestrum it was very difficult for her to enunciate clearly, and when she tried to eat food, especially liquids, they came out through the nose. Her recovery from the operation was rapid and after a few weeks there were no raw surfaces left. She was then referred to a local dentist, Dr. Wm. Mann, who made a plate that fits into the antrum and closes all the openings (see Fig. 4). This enables her to enunciate distinctly and to eat anything she likes. We have been unable to find in the literature any description of such a complete sequestrum following typhoid fever.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF OTOLOGY.

Regular Meeting, Jan. 13, 1928.

Diagnosis and Treatment of Infections of the Lateral Sinus. Dr. George Loring Tobey.

DISCUSSION.

DR. W. H. HASKIN: Naturally I agree with all that has been said by Dr. Dwyer, my associate, and I agree with a great deal of what Dr. Tobey said; though with some I do not. The question of uncovering the sinuses in all mastoid operations, I do not believe in. It may be all right in the hands of a skillful operator, but there is a grave risk. I do not believe that small injuries or spicules of bone are frequently the cause of infection in the lateral sinuses. Instead of tieing the jugular high up, we do it low down; in requires only half the time, and the results are as good as in the deeper incision and tieing that way.

Another question: Where there is undoubted thrombosis in the lateral sinus we use suction to remove the clots; then put in plugs. This will obviate the necessity of making the long bony dissection posteriorly. You get an inflow of fresh blood which clots and is more or less sterile, and so far we have not had any bad results.

We have not used the manimeter at the Manhattan. Dr. Tobey is a great advocate of it; but looking back 40 years, it is interesting to note that we used then to be able to diagnose sinus thrombosis and treat it, and our results were not much behind those of the present day. I do feel, though, that we should use every possible test—blood counts and cultures, etc.—and not wait too long. The blood count is very valuable, but after all that is said and done, the personal equation of the man who is attending the case must be considered. A man of good judgment, after long years of experience, will detect these cases time and time again where another man will sit by and waste time trying to make up his mind whether it is or is not a septic thrombosis. Many cases of infection of the sinus occur after too long periods on operating table. An ordinary mastoid operation shold not require much more than half an hour, unless there are complications; longer periods of operation reduce the stamina of the patient, and his natural resistance is being lowered all the time. In children where there is not much bone to be removed, 10 or 15 minutes should be enough, and the child will recover quickly. I don't believe that many otologists have operated on as few sinus thromboses as I. I have always made a point of rapid operation; it is not a question of shqwing off, but of giving the patient every chance; making as small an incision as possible and doing a raipd and complete operation, and see what will happen, when the sinus is exposed; pathologically it is wise to uncover it thoroughly, so that if later on you have to open it, you do not have to do further operation. But the main question is the personality of the man handling the case. You see some waiting day after day for a positive blood culture. That is folly. When you have evidence of a positive infection and operate promptly, it is a comparatively safe procedure. On the other hand, I have not operated on several children who were going along day after day with an apparently septic temperature, though I could not be convinced that they were having a thrombosis. Two or three such cases were sent to the children's hospital, and in each instance after a few days, by attending to the intestinal tract, etc., the condition subsided. Of course, the pyelitis cases have to be attended to, but intestinal cases are difficult and the pediatrician must be called in.

DR. JOHN R. PAGE: Dr. Dwyer and Dr. Tobey need not apologize for bringing up an old subject. It is one that interests everyone here, and although the hour is late everyone here would like to say something about it. An interesting point in the anatomy that was not referred to by Dr. Dwyer occurred to me when Dr. Tobey spoke of working in the upper part of the sigmoid,

that is, the entrance of the superior petrosal into the lateral sinus, which is often by several openings 3-4 m.m. apart, extending along the sinus wall 2 or more c.m. Unless this is appreciated, the hemorrhage may be embarrassing and difficult to control. The triangular portion where it lies between the layers of the tentorium makes hemorrhage harder to control in that region also than in the sigmoid region, where the cross-section is semicylindrical.

What Dr. Tobey said about ligation was very interesting. I have never performed it, perhaps more because I began by resecting, and while operating feel the patient has a better chance with the method I am more familiar with. I feel I work better and faster with resection. However, his statistics encourage me.

In simple mastoids I don't feel that it is best to expose the sinus in all cases. I used to expose it more often than I do now, for then the wounds were left more open. Now, in letting a wound close I don't want the flaps to lie on a freshly exposed sinus wall.

As for the manometer, I was with Dr. Tobey three years ago in Jacksonville when he described the use of it. I was interested and had the hospital get one on my return, but in all this time I have not had an opportunity to use it. Others at the Manhattan have used it, but some, not having read his paper, did not appreciate the detail necessary to quiet the patient during its use and were not favorably impressed. I think it is very valuable and am looking for an opportunity to use it.

DR. T. L. SAUNDERS: I wish to express my appreciation of the papers by Dr. Dwyer and Dr. Tobey, and especially to Dr. Tobey for coming to New York and giving us this very delightful and instructive paper. I think after his exposition of the manometer test we will all want to try it.

I have always felt that the clinical symptoms of sinus thrombosis are paramount in the diagnosis, and take precedence of the laboratory tests, which, however, are valuable aids. I agree with Dr. Haskins that the ultimate outcome of a case depends in no small measure upon the surgical judgment and technique of the physician in charge.

As for the question of ligation, I think that Dr. Page rather happily expressed my sentiments. There is one case in which I would probably favor litigation rather than excision, and that is in a woman, where the cosmetic result is of especial importance.

DR. G. L. TOBEY: I only wish to say that the manometer test has never been labeled with the diagnosis of sinus thrombosis; it is simply one of the aids to the clinical diagnosis.

DR. MCCOY: I have listened with interest to these papers by Dr. Dwyer and Dr. Tobey, and have much enjoyed them. They have discussed the anatomy, pathology and treatment quite thoroughly, so I will just make a few remarks on the diagnosis and treatment of sinus thrombosis as it appears to me. Looking back over my experience, I find two types of cases—the one beginning with a typical chill and fever, and sweat, and giving a positive blood culture; such a case would be described by Dr. Tobey as the hemorrhagic type, where the infection promptly gets inside of the blood vessels and finally the lateral sinus, and gives a systemic infection from the start. The other type is the neglected coalescent type, where the patient has been allowed to go along for weeks, and after two or three weeks develops typical symptoms. Such cases, however, are in the minority. The atypical cases are the ones which give us the most trouble in making a diagnosis, in that they have indefinite symptoms and show misleading signs. One of my colleagues had a patient on whose mastoid he operated. This boy developed a temperature; a blood count was taken, and he was found to have a leucopenia. A medical consultant was called in, who said the case was suggestive of typhoid. The boy was watched for a week and finally was operated on for sinus thrombosis. Had Dr. Tobey's test been applied, the case would probably have ben diagnosed. The irregular type of case bothers us. For such cases a number of tests have been devised—the Greisinger test; the Crow test, by pressing on the internal jugular vein and noting if the blood vessels of the eye dilate. I remember two cases; in both it took four minutes by the watch; it seemed a long time to make pressure, and then the enlargement of the vessels became prominent.

Then we have the test of watching the eye grounds—the dilated vessels on that side. This sometimes helps in a case of double mastoid.

Then we have the test of taking cultures from both jugular veins and counting the colonies, and assuming that the side showing the greater number of colonies is the one infected.

Then there is the test of counting the hemoglobin and red cells; if we find a constant diminution in the red cells and the hemoglobin it makes us suspect sinus thrombosis.

Then we have the test devised by Kreger, who found that in extradural abscess or in sinus thrombosis the cells in the spinal fluid run from 25 to 250; in subdural or brain abscess they run from 250 to 2,500; when higher than this, meningitis is suspected.

Thus, we see we have a number of tests to lead us out of the land of doubt into something positive. Now comes Dr. Tobey and tells us that we have the protection of this spinal fluid manometer test; that by making pressure on the normal internal jugular vein the manometer pressure rises, but if sinus thrombosis exists there is no rise. The Quackenstedt test has been used a great deal by the neurologists, and I would like to ask Dr. Tobey a few questions.

First, have most of your tests been made with the patient under total or under local anesthesia?

DR. TOBEY: Both under local and under general anesthesia; more, perhaps, under local anesthesia.

DR. MCCOY: The point I wish to bring out is that the spinal fluid pressure is doubled under general anesthesia; normal spinal fluid pressure is .8 m.m. mercury.

DR. TOBEY: Over a large series of cases, a pressure of 150 seemed to be a relatively normal pressure with the manometer which we used, namely, the Ayer manometer.

DR. MCCOY: At the New York Eye and Ear Infirmary, we have a manometer called the Strauss manometer, very similar to yours, but it begins at 5, 10, 15, 20, and goes only as high as 60. In other words, the spinal fluid is allowed to come into this tube, and I suppose it means water pressure in centimeters, rather than mercury pressure.

DR. TOBEY: The type of manometer used for this test is immaterial, since it is the relative pressure and the variation of pressure following the occlusion of the internal jugular vein on alternate sides which we desire.

DR. MCCOY: Dr. Tobey spoke of the dangers attending this test and of not using it in cases of choked disc. The neurologists say, then you will shut us off from using this test, for most of the cases have choked disc; and we prefer to use a test that requires only a small amount of fluid to come out, and that gives us a reading of .8 for normal; but they claim that with this type of manometer there is danger of letting too much spinal fluid in the tube and getting into the danger zone of possible sudden death.

DR. TOBEY: We mentioned the problematical loss of a relatively large amount of fluid and suggested the possible danger in the presence of a subtentorial tumor.

DR. MCCOY: We use the Strauss manometer, and have found that if the patient coughs or moves it makes the pressure vary up or down, and we feel we are not justified in making a positive diagnosis. We feel that Dr. Tobey's sign is something similar to transillumination—if positive, it is a very good sign; if doubtful, one had better rely on other things.

In the treatment of these conditions, I think the type of case, which is very difficult, is where you find a tremendous amount of fibrin spreads through the neck and it is difficult to locate the vessels, and sometimes you will find that you lose the vein, for it is almost strangled in the fibrin thrown out. That is a type of case that has to be considered. In one such case I had to use the gland at the angle of the jaw as my guide; I could not feel the carotid. I went into the lumen of the vein, and the vein was completely collapsed. In such a case the transverse incision would be more difficult than the long incision.

Then I have had other cases. In one instance I operated on both sinuses of a boy. I had to go away, and I had someone else dress him, with the result that at the first dressing the doctor opened one sinus when dressing it, and some days later opened the other side when dressing it—and when I got back two weeks later I found the patient going on very well. Apparently it had no effect on him.

At one time I reported a case of erysipelas intervening. The patient was having a jugular resection done, which took a long time, and he suddenly went into a collapse and was put to bed; the next day he was found to have erysipelas, and being a ward patient, he was sent to Bellevue, where nothing was done to the sinus thrombosis, and yet the patient went on to recovery.

I feel that we are greatly indebted to Dr. Tobey for coming here and presenting this paper. It is always a pleasure to see and to hear him.

SECTION OF OTOLOGY.

Regular Meeting, Feb. 10, 1928.

Two Cases of Petrous Bone Abscess Drainage; Recovery. Dr. Wesley C. Bowers.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Report of Case, with Pathological Specimen. Dr. Isidore Friesner.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Report of a Case of Suppurative Osteitis of the Petrous Pyramid. Dr. Edw. L. Pratt.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

Suppuration of the Petrous Pyramid, etc. Dr. R. T. Atkins.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. FOSTER KENNEDY: I have seen only two cases where I have known of this condition, so my experience is small and I do not feel able to say that I could diagnose a third case with any kind of certainty from the experience I had with these two. I was interested in Dr. Eagleton's statement that if we collected the signs of meningitis in the middle and posterior fossae we could make a diagnosis, but I am not optimistic about that. After all, nearly all the basal meningitides will produce a middle and posterior syndrome.

In my second case, where the initial lesion was a bullet inside the petrous bone, which produced fifth nerve neuralgia, for which he came in 18 months after he had received the bullet shot, which was still in position—he was having so much pain in his fifth nerve area that it seemed wise to remove the bullet, but we were very shy of doing it, for he gave a history of three separate attacks of severe meningitis during the 18 months, from which he had recovered. He was a very intelligent person and said he had been lumbar punctured and pus cells were found in his fluid; after the removal of the fluid he was relieved of his fifth nerve neuralgia, but he had afterwards a succession of attacks of meningitis, with intervals of complete well being. During the attacks of meningitis he had a very high cell count—many hundreds of cells, with stiff neck, and Kernig's sign, etc. In two of them it was thought he was going to die, but he recovered. Finally, however, he had an attack from which he did not recover. But he recovered from three separate attacks of meningitis which to our clinical eyes we believed would be fatal. That was the only time I have seen successive attacks of septic meningitis apparently recover.

When the autopsy was done he was found to have complete suppuration of not just the apex but the entire two-thirds of the pyramid, and the petrous bone was saturated.

In the case that Dr. Atkins spoke of, we always strenuously believed he did not have a brain abscess, and went on that faith. He began with Gradenigo's syndrome, which he recovered from, but that is often a benign situation, the result of edema. There developed a condition of great headache and a tremendous leucocyte count. He went up to 85 or 90 per cent polys., and we thought he had a sinus thrombosis. Dr. Taylor operated and failed to find any abscess; but we thought he had a localized meningitis with a fistula. We thought the leucocyte count meant sinus thrombosis, but tieing off the jugular was not conclusive.

I was interested to hear Dr. Eagleton say that two cases of recovery had been reported, for I did not believe that this condition could be recovered from. They must have been in the very early stages, for when the petrous bone and

the texture becomes affected it seems extraordinary to be able to remove it without subsequent infection. The diagnosis must have been made as a tour de force very early, for in the two cases I have seen I don't think any surgical operation could have saved the patients.

DR. ALFRED S. TAYLOR: My experience has been limited to the same two cases that Dr. Kennedy has spoken of, so that my evening has been more valuable to me than my remarks to you.

I was particularly interested in Dr. Friesner's explanation of the extension of the ear disturbance to the petrous pyramid. I should think that extension probably occurred in our first case, for in the exploration the dura had been lifted from the posterior surface as far as the internal meatus, and the tissues were normal. The dura had also been elevated from the anterior surface as far as the Gasserian ganglion and there also it looked normal, so the direct infection must have gone from the middle or internal ear through the petrous bone to where the carotid comes up through.

In watching the autopsy in that case, it was very interesting to see that the petrous bone on the surface looked normal until we got to the tip and there one could see a thin pus exuding from the place where the carotid went through, and a little perforation there of the dura into the arachnoid space and into the third ventricle. My recollection is that the thing that caused the sudden death had been a perforation into the floor of the third ventricle, the contents of which were decidedly purulent. The boy had been feeling fairly well on Sunday morning, had sat up and asked for a drink of water, which was given to him, and he suddenly fell back on his pillow and was dead in half an hour. I thought that the perforation of the abscess into the ventricle was the reason of his sudden collapse.

In the other case, that of the man with the bullet, the bullet started in the cheek and went through the nasal fossa, through the sphenoid bone and the middle fossa, and lodged in the tip of the right petrous bone. It was a .38 calibre, ½-inch long, and when we went for it only the base was to be seen, and that was flush with the anterior surface of the pertous. It had cut the petrous into two fragments, the upper of which elevated the dura. The bone did not look particularly affected and I hoped we could get away with a good recovery. He had three postoperative attacks of meningitis, and had had several attacks in the preceding year. During these attacks, the infusion of hypertonic glucose solution into the vein would cause a drop of temperature and relieve him in a few hours. During the dressing of his wound, which showed some discharge, we used Dakin's solution and he complained that the Dakin solution tasted very disagreeably. I did not think he could really taste it, but at the autopsy a continuous sinus from his nose through the middle fossa was found, so that the prospect was hopeless.

Concerning the wisdom of going to the tip of the petrous subdurally, it would seem that if the dura were elevated from the anterior surface, one would have to elevate the fifth nerve ganglion, and under the ganglion there are always veins which bleed much, and if an abscess is found one can get no good drainage because of the pressure downward of the dura. A large enough drain or a firm enough tube would damage the brain structures above the dura. Such drainage would have to continue over a long period of time—perhaps months. I cannot feel, considering the conditions, as Dr. Eagleton seems to feel, that a good proportion of them ought to get well; it would be in the nature of a miracle. One could not approach along the posterior surface, as there would almost surely arise an infection within the dura. Here again the drainage would be continued for so long that one would be almost sure to get damage of the brain. It would therefore seem that if one were to get any approach it would have to follow the line of progress of the infection. As was said by Dr. Friesner, it would have to go within the petrous pyramid; and that would be the logical way of approach, for if you could get your canal through the bone you could drain more or less indefinitely and you would have the cortex of the petrous for protection. That would be better than elevating the dura, for in many procedures that does not work out satisfactorily.

Concerning the sixth nerve palsy which recovered after the laminectomy, I think we should remember that the sixth nerve is very susceptible to loss of function from increased intracranial pressure, etc. This is explained by many on the basis that the sixth nerve, as it passes by the middle fossa and goes

along the sphenoid, is flanked by a little bony hook, and if the tension is greatly increased the nerve is likely to be pressed on the hook and become paralyzed. The history was that there was considerable increase of pressure in this instance. I should think that the palsy was partly due to the increased intracranial pressure, and this pressure was relieved by the laminectomy and drainage.

Another point that interested me was the fact that in most of the cases reported pain back of the eye was one of the early end trouble symptoms. The innermost fibres of the posterior root of the fifth nerve supply the ophthalmic branch with sensory fibres, and if the pains back of the eye are one of the early symptoms in a given case, then one is probably dealing with an infection of the tip of the petrous with irritation of the ophthalmic portion of the root, and I would consider that sign as a strong indication, if one were going to operate, to get at it soon.

I was also interested to hear that these infections would cause a local meningitis, but my impression was that if the infection had gotten into the subarachnoid space it would no longer be localized. These infections do wall off, as do many of the other types of infection, and if these cases are to be cured while in the stage of local meningitis it would be only while there is a pachymeningitis rather than a true subarachnoid infection.

DR. PRATT: I certainly cannot *add* anything to what has already been so admirably expressed by those who have had a far greater clinical experience than I have had. *My* experience is even more limited than that of any of the previous speakers, for I have had but *one* case of suppurative involvement of the petrous pyramid.

I must confess, however, that the discussion of the cases reported tonight has left me somewhat perplexed. I am perplexed by Dr. Eagleton's statement that these cases have "a definite symptom complex" and that "they are amenable to operation". It seems to me, from my very limited clinical experience and from a rather superficial review of the literature, that the symptoms in these cases *may be*, and not infrequently *are*, most diversified. The symptoms, of course, are dependent upon the pathology. If the pathological process in the petrous pyramid is so situated as to involve the fifth nerve, or the sixth nerve, or both nerves, there is naturally a definite symptoms complex present. My case had no such symptom complex and the autopsy failed to reveal any pathology associated with either the fifth or the sixth nerve. The first symptom in my case which gave any clue to a diagnosis was really not a symptom but a physical sign—rigidity of the neck. In my case there were no symptoms and no physical signs *throughout the entire course of the disease* of either fifth or sixth nerve involvement.

Dr. Eagleton advocates early operation in these cases. He cited two cases which had recovered following operation. One of the previous speakers stated —if I hard correctly—that he considered the recovery in these two cases to be more in the nature of a miracle than a surgical achievement, and I must confess that I agree with this opinion. Early operation *may* be a justifiable procedure if a definite symptom complex gives one a clue to the pathology. Certainly in the case which I have reported there were neither symptoms nor physical signs to justify an "early" exploration of the petrous pyramid.

There have been many references to the significance of fifth and sixth nerve paresis or paralysis as a symptom in these cases. My case had no such symptoms and the autopsy showed that it is possible to have a suppurative osteitis of the petrous pyramid so limited as to cause no involvement of either the fifth or the sixth nerves.

I agree with Dr. Kennedy that a sixth nerve paralysis associated with a suppurative condition of the middle ear or the mastoid process does not necessarily mean that we are dealing with a localized meningitis caused by a suppurative process in the petrous portion of the temporal bone.

I had a case of sixth nerve paralysis following a simple mastoidotomy about a year ago, at which your Chairman came to my assistance in consultation. The typical Gradenigo syndrome developed 15 days after a simple mastoidotomy. Although there were no positive signs of sinus thrombosis, it was the opinion of two consultants that the sigmoid sinus should be explored. This was done. The internal jugular vein was ligated and the sigmoid sinus exposed and opened. Free bleeding took place and no clot was found. The next morning (about 12 hours after the operation) the temporofrontal headache disappeared, never

recurred and the external rectus muscle gradually recovered its normal function.

The sudden relief from headache following the ligation of the jugular and the exploration of the lateral sinus in this case appeared to me to justify the conclusion that such a sudden relief could never have taken place if the sixth nerve paralysis was caused by an inflammatory process of the petrous pyramid, or by a localized meningitis.

I confess that my clinical experience in these cases is very limited, but I am still perplexed by the problem of *when* to adopt surgical measures, and what *type* of operation offers the most hope of successful results in cases of suppurative involvement of the petrous pyramid.

DR. EAGLETON: Died of suppurative meningitis?

DR. FRIESNER: He did not. I do feel that the pathway of attack should be through the middle fossa, for the reason that so many get well. It is a pity to destroy the ear in an attempt to reach the focus through a narrow gorge; it is possible, but it is dangerous and should be avoided if possible.

SECTION OF LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, Feb. 23, 1928.

Rhinolith Requiring External Nasal Operation for Its Removal. Dr. M. C. Myerson.

(*To appear in a subsequent issue of* THE LARYNGOSCOPE.)

Foreign Body In Nose. Dr. M. C. Myerson.

(*To appear in a subsequent issue of* THE LARYNGOSCOPE.)

Improved Anesthesia for Antrum Puncture, etc. Dr. M. C. Myerson.

(*To appear in a subsequent issue of* THE LARYNGOSCOPE.)

DISCUSSION.

DR. HARMON SMITH: In his operation on the teratoma case, Dr. Lore was very fortunate in not having the hemorrhage to be anticipated in such cases. The borderline of malignity is so close in territoma, however, that I fear he cannot lay mueh unction to his soul in regard to a permanent cure, for the patient will, in all probability, have a recurrence.

Some years ago I reported three cases of lymphosarcoma of the tonsils. They are the least malignant of any of the malignant tumors that invade the tonsillar tissue, so we may feel that he has more reason to expect a satisfactory result than from the removal of any other malignant tumor of the mucous membrane. However, that does not mean that one will not get a recurrence in some other place; as a metastasis following the removal of these tumors of the tonsil takes place almost invariably, and sometimes very remotely from the site of the tumor removed. In one sarcoma case I removed the tonsil of one side successfully and without bleeding, but there was recurrence on the other side, which was also successfully removed. Then it recurred in the brain, associated with delirium and followed by death. The second case had a recurrence in the liver. In none of the cases did the disease recur at the original site. That was long before we employed radium, light, heat, fulguration, etc., which we now have at command. With the various measures we now can employ, we may be able to operate successfully and prevent metastases.

In the third case, I agree with Dr. Lore that nature has performed a much more successful radical removal than he or anyone else could have done. The flaps undoubtedly can be transplanted so as to cover the defect near the nose; they are sufficiently vital, and if there is no recurrence of the malignity there is no reason why he should not expect proper union of the flap.

DR. FRANCIS W. WHITE: I remember the first case (teratoma) very well indeed, and I congratulate Dr. Lore on the result, whether or not there is a recurrence later. It was a beautiful piece of work. I don't know why he invited me to assist him, but it was well worth while to be there. I have seen a lot of work done in the nasopharynx, but have never seen better. In sincerely hope the patient will not have a recurrence.

Bloodless Tonsil Enucleation. Dr. F. Peter Herman.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. R. H. FOWLER: It is an honor to be asked to discuss Dr. Herman's paper.

I have seen the instrument tried out at Manhattan during the last few days by Dr. Harmon Smith and Dr. Francis White. Both of these gentlemen are here and it seems fitting that I leave to them the task of discussing the ease or difficulty in using it or whether they were pleased with the results obtained.

For my part, if you will allow me to show four slides I shall attempt to take up the more fundamental question; that is, the physical problems to be overcome in the enucleation of the tonsil by whatever method is used.

There is a reason—an anatomical reason—why, up to the present, attempts to accomplish this with a one-instrument procedure have been disappointing.

Athwart the tonsil lies the shadow of a mysterious muscle which attaches to the capsule. The understanding of this little known structure and its handling is the crux of the matter.

In the more modern operation of open dissection enucleation of the upper lobe is a comparatively simple proceeding. The field is so dry and visible that the surgeon as he strips the tonsil from its bed can see a mass of muscle tissue attached to the capsule throughout the anteroposterior extent of the tonsil, somewhat below its middle. In fact, at the groove which marks the junction of the upper and lower lobe.

If the snare be used at this point in the operation it is apt to damage the muscle and cause a bleeding; but, on the contrary, if this muscle be teased away, that is to say, freed by open dissection exactly at the line of its attachment, complete enucleation can be carried through.

This step is a new feature that has been added to the orthodox operation in the last five years. It makes for less bleeding because, as you see in this slide, the vessels that lie close to the capsule in the smaller branches. It makes for less trauma because it saves the muscle and pharyngeal fascia it covers and it permits the more accurate removal of the lower lobe by revealing it to direct vision and freeing its most important attachments. With this forward step called carrying dissection to the base, the orthodox operation has been greatly advanced. So much for the operation done by one school.

Another school of laryngologists has made strenuous efforts to perfect the use of automatic instruments. It must be acknowledged that they have made great strides and have overcome many of the mechanical difficulties. This evening Dr. Herman has shown us the latest example. Taking up these automatic instruments, as a group, it may fairly be said that they will finally be judged on the same standards as open surgical dissection, that is, on the basis of results and of adaption to anatomical conditions.

From what I have seen, it can fairly be classed with the other single instruments that have been put forth with similar claims for recognition as to the solution of the problem. This procedure, as Dr. Herman has said, is dependent upon the operator being familiar with the Beck-Schenk technique and, as Dr. Nilson, who has tried it out, tells me, the results are closely similar to those obtained with that type of instrument, the difference being only the addition of high frequency current in the wire. In all other respects it is essentially the same.

What then are the results? Excellent in children. Not so satisfactory in adults or where scars are present.

And again what shall be said of the adaptability of automatic instruments to the anatomical conditions?

They are well adapted to the infantile type of tonsil shown in the upper part of this slide. Here the muscle is slight and the groove is shallow, thus favoring the automatic action of the wire.

On the contrary, they are not adapted to the adult type, where the muscle is well developed and the groove to which it attaches is deep.

In short, it remains for you surgeons, with these physical conditions kept clearly in mind, to decide whether your own trained eye and hand will or will not discriminate more accurately between the various anatomical structures, rather than the unseen wire or blade guided by a ring.

DR. HARMON SMITH: We are deeply indebted to Dr. Herman for coming from Florida and presenting this subject to us in this very modest way. His number of cases qualifies him to speak advisedly. The small number of cases that I have had opportunity to do or see is not sufficient for judging the method. My experiences bear no relation whatever to the effectiveness of the operation, for the same reason that when Sluder presented his method of procedure many years ago, many of us took exceptions to it; but when he came to New York and demonstrated on 20 or 30 cases at the Manhattan, we all recognized that there was great virtue in it, particularly in children. Now, my objections to this procedure may be based on similar grounds—lack of proper technique. I believe that with proper instruction from an adept, the method would be effective and would have many advantages. I have seen only a few cases and those few "swallows" do not make a summer. In one case I operated upon the result was most satisfactory. In the second case, the tonsils were too large to be engaged in the instrument and when I used the wire snare alone the wire broke. I rather favor the wire loop than the instrument with the fenestra, for the fenestra is limited in its size to accommodate itself to varying sized tonsils. The tonsils were removed under local anesthesia. Possibly I made the error of trying to separate the tonsil from the capsule before engaging them in the instrument. Whether we can employ an instrument of this kind, as in the Sluder method, by pressing it up against the posterior border of the alveola process, I don't know. I imagine that under ether it would be more satisfactory. In using the needle, I found it was too short, and I lost my way in endeavoring to separate the folds, and did not know whether I had sufficiently separated them or not. Third, I had a short circuit, which shocked me considerably, and I approached the next subject with some trepidation, but, operating on more cases and with Dr. Herman to guide me, I may assume greater fortitude. The next day there was the usual trauma of the tissues and edema. Whether that was due to poor technique or to the instrument, I don't know. However, I have gotten the same conditions with the cold snare. None of these cases had a subsequent hemorrhage, and there was a clean capsule remaining. I wish to extend to Dr. Herman the privilege of coming to the Manhattan Hospital to demonstrate on a number of cases the method that he employs and then he will be able to accept or reject, beyond question, what he has said in regard to it.

DR. WHITE: I wish to add my word of thanks to Dr. Herman for coming here and demonstrating this new procedure to us. Anything I may have to say is not in the nature of criticism of Dr. Herman or his advocacy of this new method, but in the way in which he did it. I saw Dr. Smith perform two operations and then I did two and came to a certain number of conclusions that I am asking myself for solutions I hope will be answered by Dr. Herman.

1. If it is necessary to do extensive separation, the method has no particular advantages.

2. In any instance where there is a shorter route than around the outside of the capsule, the burning wire will take it, thus leaving a stump, which must of necessity be removed later.

3. The reaction after the operation is about the same as in those done by other methods.

4. That any operative procedure requiring an inordinate number of instruments or complicated mechanisms is not destined to become popular.

5. No new method should be accepted or condemned until a number of operators have done a considerable number of operations under varied circumstances.

6. In the event of the wire breaking while the current is on, could a short circuit be established, thus producing a spark and igniting the ether? I saw Dr. Smith get a shock and I can still show the result of a burn on one of my fingers. If the tonsillar fossae of the patients operated upon are as sore as my finger, they are certainly having some pain tonight!

7. Oftentime, after the use of the cold snare, a large blood vessel may be seen protruding into a tonsillar fossa—what is the effect of this particular method upon such a vessel?

8. Should the passage of the wire be rapid or slow after turning on the electric current?

9. I am certain that after a little experience, my technique would be more co-ordinated and thus improved, for the loss of blood in these patients was more than usual than when using my regular technique.

I hope Dr. Herman will accept Dr. Smith's invitation to come to the Manhattan Eye, Ear and Throat Hospital and perform a few operations. It so happened that both of my operations were in patients with badly diseased fibroid tonsils and I probably would have had some difficulty in removing them with the ordinary cold snare, and I am anxious to see Dr. Herman's method in detail.

DR. ARTHUR NILSEN: I have done 30 or 40 cases with this instrument, using, however, Dr. Herman's earlier techinque, with the dispersing electrode attached to a large clamp around the patient's thigh. With this method the current passes from the wire through the patient's body and produces probably more slough of the tonsil fossa than the latest technique. I experienced no particular difficulties of any kind, for one reason because I am familiar with the Beck-Mueller technique, and anyone not familiar with this technique will have trouble with this instrument. The dislocation and engagement of the tonsil with this instrument is identical with the use of the Beck-Mueller snare.

I can answer two of Dr. White's questions; first, the necessity of preliminary separating. There is no preliminary separating in the Beck-Mueller technique. The tonsil is properly dislocated through the ring and the wire tightly engaged, following the line of cleavage. Second, the current is not turned on until the wire is pulled tight and is in the proper line of cleavage. Thus, the electrified wire simply follows through outside the capsule and does not cut through the tonsil and leave a stump.

The Beck-Mueller method is not suited for all types of tonsil. There are some so adherent that you cannot get them with any of the so-called mechanical dislocation instruments, such as the Beck-Mueller, Sluder or Braun, but they must be dissected. The vast majority of tonsils, however, can be removed with the Beck-Mueller instrument, either cold wire or electrified, if the operator is familiar with the technique.

I had one secondary hemorrhage in an adult, perhaps because I used too much current and had too much slough, although it did not appear excessive. On the seventh day the patient was packing to leave town and ran up three flights of stairs, starting a hemorrhage. I have done a number of cases using the cold wire snare on one tonsil and the electrified wire on the other, and compared them until complete healing took place. There seemed to be little if any difference in the appearance of the two sides at any stage, in spite of the fact that I used the old techinque and probably more current than was necessary, except of course that the electrified side was a completely bloodless operation.

I believe, however, that the technique which Dr. Herman has just described, with the dispersing electrode attached to the tenaculum, is better than the method I have used, in that it produces less slough and consequently prompter healing with less risk of secondary bleeding; nevertheless, my experience with this instrument has been most satisfactory.

DR. ALFRED BRAUN: I think the method described has a certain amount of merit, especially in the case of hemophiliacs, where the question of bleeding is a predominant factor. In such cases the method would seem to be particularly useful, although even here I imagine there might be secondary bleeding, as in every type of operation if enough of these are done. The one objection I see is the unecessary multiplication of instruments, and the possible difficulty in the management of the currents; the possibilty of the operator having to manipulate currents and so involving his own technique in difficulty, and also the inherent difficulty in the Beck-Mueller snare. I think the instrument is a much better instrument for the dislocation of the tonsil, etc. That is why I believe the adopting of this same type of instrument, or electrical instrument might be a little more effective. However that is not the subject. I believe that in a large majority of cases the instrument we have and the proper technique developed with that particular type of instrument will give a result which will preclude the necessity of using an instrument which is a little difficult.

(To be continued.)

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Meeting of February 7, 1928.

COLLEGE OF PHYSICIANS.

Five Mastoidectomies and a Decompression Operation On the Same Patient. Dr. Philip S. Stout.

DISCUSSION.

DR. RALPH BUTLER: Dr. Stout's case brings to my mind a patient whom we treated at the University Hospital with Dr. B. Alexander Randall. She had been operated on several times before we saw her and was having severe pain about the ear. Dr. Randall said the ear condition was tubercular and eventually a slight pulmonary tuberculosis was found. The mastoid cleared up without further operation.

DR. GEORGE E. ALEXANDER: It is not clear to me how a simple mastoid operation can follow five radical ones on same mastoid process and ear. The essayist may be referring to a minor or modified operation in the mastoid region for the removal of a small area of necrotic tissues, etc., existing subsequent to the previous operations. I would therefore ask him to please explain more fully the conditions found and the technique of the last or simple mastoid operation.

DR. HENRY S. WIEDER: This case brings to my mind a patient at the Polyclinic Hospital who had three or four mastoid operations. At the last operation I cleaned out absolutely everything and he still complained of pain over the mastoid area. We discovered that the cause of this was due to a Meckel's ganglion neuralgia, the pain stopping immediately when cocain was applied to the ganglion.

DR. STOUT, closing discussion: In answer to the question why we did not do a complete radical mastoid in the beginning, would say that she had considerable hearing in the right ear and a modified radical was thought best at that time. However, an almost complete radical was done later at the other hospitals, leaving only a portion of scarred drumhead.

Carcinoma Affecting the Sinuses in a Young Patient. Dr. Harry A. Schatz.

A colored girl, age 15 years, service of Dr. Butler. History of nasal obstruction for about two years, which had become worse about four months prior to her first visit to our clinic.

There are three or four factors, beside the youth of the patient, which are worthy of note. One is that a biopsy, to be reliable, must be done on a specimen that includes the suspected tissue. In this particular case a piece of the suspicious tissue was removed from the pharyngeal vault where it projected from the postnasal space. This was done at one of her earliest visits, but the assistant in Dr. Butler's Clinic who attended to this apparently did not take a sufficient amount, for the report came back, "tissue presenting chronic inflammatory character, not neoplasm".

Within a week a Wassermann test was done, the result being strongly positive. That was another factor that postponed the correct diagnosis. Bismuth injections were instituted in the Dermatological Service, some nine injections being given, yet the lesion seemed to progress, her vision began to fail in the right eye—the side of nasal involvement—some pain developed, some puffiness of the eyelids, and some protrusion of the eyeball; and X-ray showed cloudiness of all the sinuses on the affected side except the frontal.

In view of all these findings and the inefficacy of the antiluetic treatment, prompt operation was decided upon. The vision at this time was practically nil in the right eye.

The sphenoid sinus was completely opened by removal of the anterior wall, the adjacent posterior ethmoids being at the same time opened, and a large opening made into the antrum under the inferior turbinate. In spite of the careful local anesthesia and the usual employment of adrenalin, the bleeding was unusually great, and aroused the comment that only neoplasm could produce it. The operation was done largely by sense of touch.

The unusual degree of hemorrhage when this patient was first gently examined with a cotton-tipped probe to feel the extent of the nasal lesion, again the hemorrhage during operation, the speedy recurrence of indurated tissue after

operation, all pointed to neoplasm, which diagnosis was confirmed by the laboratory some weeks later when a cervical gland on the left side (not the side of her lesion) became enlarged and was removed for biopsy. The report came back, "metastatic carcinoma". X-ray treatment was then promptly instituted, and the patient left entirely in the care of the radiologists.

The mass occluding the posterior nares, the headache, the loss of vision, the nasal obstruction, all are just as bad as prior to operation.

DISCUSSION.

DR. HENRY S. WIEDER: This case is particularly interesting to me because of the age of the patient. I have always been interested in pathological specimens of carcinoma. In 1907, when I went to Medico-Chi we searched the museum trying to get specimens for teaching purposes. We could only get two superior maxillas, both taken from children; one of only 7½ years, one of which proved microscopically to be carcinoma. This was the youngest case of true carcinoma I have ever encountered. I have also seen carcinoma of the breast in a young woman age 21 years.

DR. RALPH BUTLER: We did not see this case until last fall. The diagnosis really should have been made earlier but the positive Wassermann threw us off the track and the first two or three biopsies were negative. I recall one other case of carcinoma in a young patient. This occurred in a boy age 20 years. The ethmoids were badly involved and it proved fatal. It was an inoperable case, just as this one is.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

Meeting of January 16, 1928.

An Unusual Rupture of the Eardrum. Dr. W. W. Wilkerson.

Dr. Wilkerson reported the case of Mrs. C. R. S., white, age 22 years, who was sent to him complaining of pain in her right ear.

History: Eight days previous she contracted a cold which gave her little or no trouble. Three days later she blew her nose rather gently and had a peculiar sensation in her right ear. She explained to me that her doctor told her always to blow the nose gently when she had a cold and she was very careful to follow his instructions. The peculiar sensation which she complained of in her right ear was described by her as "something having opened up". Following this occurrence she had some pain in her ear; three hours later the pain became severe. A local physician was called in. He found the ear canal filled with blood and clots of cerumen. He put her on simple treatments consisting of irrigations and phenol drops. Prior to this time the patient had had no pain in either ear, and had never suffered with earache even as a child. Five days after the rupturing of the drum she had only a slight amount of pain and some deafness in the right ear.

Examination: Both drums were markedly retracted, and a small, rough perforation was noted in the lower quadrant of the right drum. The nose and throat examination was negative. According to the history which she and her local physician gave the patient never had had a purulent secretion from her right ear.

DISCUSSION.

DR. FRED HASTY said that he had seen five unusual cases where the eardrum had been ruptured. One drum was ruptured during an inflation, another while a woman was standing on the street a manhole blew up and hit her, making her dizzy and ruptured the drum, still another drum was ruptured by a streak of lightning, and lastly a ruptured drum caused by a lick from a clothes brush.

DR. WILKERSON, in closing, said that his patient was very intelligent and that he was sure the history is correct. I haven't had time to look up the literature in connection with it. The unique feature in the case I had is the fact that the eardurm ruptured practically without pain. I don't quite understand it. I don't understand why the patient didn't have some infection, because the ear had been irrigated for five days when I saw her. I glanced through some books I had about the time I saw this case, but I could find no mention of this unusual occurrence. I shall investigate them further.